IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF TEXAS
MARSHALL DIVISION

CENTOCOR, ET AL	*	Civil Docket No.
	*	2:07-CV-139
VS.	*	Marshall, Texas
	*	
	*	June 22, 2009
ABBOTT LABORATORIES	*	8:00 A.M.

TRANSCRIPT OF TRIAL PROCEEDINGS
BEFORE THE HONORABLE JUDGE T. JOHN WARD
UNITED STATES DISTRICT JUDGE
AND A JURY

APPEARANCES:

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(Proceedings recorded by mechanical stenography, transcript produced on CAT system.)

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P R O C E E D I N G S

(Jury out.)

COURT SECURITY OFFICER: All rise.

THE COURT: Please be seated.

Good morning, counsel.

Okay. I understand y'all have a motion -- additional motion in limine or something you want to talk to me about.

MS. ELDERKIN: Yes, Your Honor, Diane Elderkin for the Plaintiffs. We have a paper copy, but perhaps I could just make this orally.

It became apparent, as we saw these opening demonstratives that Abbott wants to use, that they may be trying to make an argument that Abbott's inventors were the first to invent human anti-TNF antibodies according to our claims.

Abbott, at one point, had 102(g) prior invention defense. They put that in their invalidity contention. They never pursued it. Their expert didn't render an opinion on it, and they don't have the witness here -- witnesses here that could make or corroborate that type of defense.

Although we recognize that they're going to try to argue that the Salfeld patent is prior art and that that type of evidence should come in, we don't believe it's appropriate for them to try to imply to the jury that the Abbott inventors were the first to invent the antibodies that are covered by our patent.

And we're asking for a ruling from Your Honor to preclude them from doing that.

THE COURT: Well, your -- but the basis of your ruling (sic) is, is saying that they abandoned that defense at some point, when initially they employed it, and then they dropped it?

MS. ELDERKIN: Correct, Your Honor.

THE COURT: And they have not ever

identified an expert to present that.

Is that what you're saying?

MS. ELDERKIN: Yes, Your Honor.

THE COURT: All right. What's the Defendants' position on that?

MR. LEE: Well, Your Honor, Bill Lee. I told Ms. Elderkin this morning we're not pursuing the 102(g) defense, but the question of whether --

THE COURT: Well then, if you're not pursuing it -- what she's saying is it would be unfair to let this evidence in.

MR. LEE: No. But, Your Honor, it's relevant for four other reasons, one of which you've ruled on already.

We argue that the in limine stage that are patents, which specifically identifies e37 or Humira in the claims would come in because it was prior art, and the question of when that work got done that led to the patent would be relevant under Your Honor's in limine ruling.

The second thing is that because there is an enablement defense, which requires the jury to determine whether the patent could be practiced without undue experimentation, the question of the people who did the work, when they did it, who was first, how much

work they had to do is relevant to that issue.

And as we argued in the in limine, Your Honor, it's also relevant to willfulness, and it's also relevant to the license defense that they tend to offer -- they tend -- the license issues that are relevant to the issue of damages.

So while we did say we're not pursuing the 102(g) defense, and I said to Ms. Elderkin that we're not this morning, this evidence of when we did our work, how hard it was, who was first is still relevant to all four of those issues.

And that was specifically addressed in connection with the motion in limine on the patent, Your Honor.

THE COURT: What do you say, Ms. Elderkin?

MS. ELDERKIN: Your Honor, I don't disagree that the evidence of what they did and how hard it may have been probably is relevant to enablement, but in terms of who did it first, I don't believe that is at all relevant.

THE COURT: That's a matter of fact as to when they did it, so that's before y'all -- before your side did it or before your client did it, then your motion in limine is denied.

Now then, there's a pending motion in limine with respect to the witness -- Murphy, I believe, his name is -- that was going to -- concerning the Defendants filed a motion in limine; is that right?

MR. LEE: That's correct, Your Honor.

THE COURT: Well, the Court's looked at that. I'm inclined to deny it as long as -- because I understand the Plaintiffs' position is that he's going to just testify about the prosecution history as revealed in his expert report, and they're not going to offer -- as I understand their briefing, they're not going to offer anything from Murphy about invalidity, rebuttal on that.

Is that correct? Ms. Elderkin, did I misread it?

MS. ELDERKIN: You haven't misread it, Your Honor. It's purely to talk about the prosecution history. He said plainly in his report that one of the things he might testify about is the prosecution history.

And he actually described it in an exhibit to his report, which I think was not submitted to the Court. It has a detailed description. He's not going to be offering any opinions, any statements of law; just what happened in the prosecution history.

THE COURT: Well, I'm denying the motion in limine. I -- you know, we still entertain objections on your feet in this Court. We haven't -- I have not restricted those.

The Court's got one other matter that was raised, I guess in the Plaintiffs' response to your motion -- to the motion in limine that raises the issue of estoppel.

Exactly what are you saying, Ms. Elderkin? Do you recall that that was in one of your replies or one of your pleadings?

MS. ELDERKIN: Yes, Your Honor. It had to do with the issue of judicial estoppel.

Abbott propounded in its motion for summary judgment on the acquiescence issue that you granted, that objections had -- that objections had been made to the 1992 application, because it wasn't enabling, and that Centocor had basically acceded to that by adding things to that application, and they should be estopped now judicially, because they prevailed on that motion.

And your opinion adopted some of those same arguments. They should be precluded now under judicial estoppel from taking a contrary position.

THE COURT: What do you say about that

one, Mr. Lee?

MR. LEE: I'm sorry, Your Honor.

THE COURT: I understand. We're a little tight in here.

I'll tell you, I've been on the -- one of these committees, you know. This courtroom is about 8 foot more narrow than the suggested guidelines. So this is what we've got.

MR. LEE: I'm going to try not to knock Mr. Beck over.

THE COURT: That's all right. You have permission.

MR. LEE: Your Honor, I'm not sure what she's asking you to estoppel us from saying. We can't be estopped from challenging enablement of the 1994 patent, because all that occurs under Your Honor's acquiescence opinion is that they are estopped from relying upon an earlier date.

The patent gets the filing date of '94. It gets a presumption of validity, but then we're entitled to attack enablement --

THE COURT: I think where she's going is not saying for the -- when the application was originally filed, was that '91?

MS. ELDERKIN: '91, and then there was

this '92 application.

THE COURT: Okay. Where she's going is to estoppel you from claiming that those filings were anticipatory of the '94 filing.

MR. LEE: And, Your Honor, I think -- I think that --

THE COURT: Am I correct in what you're --

MS. ELDERKIN: Yes.

THE COURT: That and the foreign application that was filed simultaneously.

MR. LEE: Right.

THE COURT: Those -- that's where we're headed.

MR. LEE: I think we're collapsing two issues. If I could just have a minute, I think I can break them down.

Your Honor, in the acquiescence ruling, the only question was, what did the Examiner say? Was there an enablement objection? Did we acquiesce? And that is a legal question that Your Honor has ruled on, and it has consequences for the priority date.

Once we get to the second set of issues, which is you have the 1994 priority date. The 1992 published application is now, indisputably, prior art to

that. It was published 18 months before the 1994 date. What it discloses it discloses, right? And if it anticipates --

THE COURT: There's no question it's admissible as to obviousness, okay?

MR. LEE: Right.

THE COURT: But I think her -- the argument is, is the question of whether your expert should be entitled to express an opinion that those earlier applications were anticipatory. They rely on one publication -- well, you know, anticipatory -- you know it better than I do. See, I can't even say it. You know it much better than I do.

Everybody in this courtroom knows that, so you know what I'm talking about.

MR. LEE: Actually, Your Honor, I think what they're saying is we're estopped from having our expert take the position that those -- the 1992 reference is enabled, not just anticipatory, I think.

MS. ELDERKIN: No. Part of anticipation -- for it to be an anticipatory, it has to be --

THE COURT: Well, that's what I said. That's the issue.

MR. LEE: Yeah. And I think, Your Honor,

this is one of the things we've suggested we could take up at a break. It's not going to come up in the openings, but I think we can show you in our expert report precisely what our expert said, which is he said the following: He said, I don't believe that it's enablement, but if their expert is correct and it's enabled, then it anticipates.

And we'll show you the paragraphs before we ever offer it to the jury. We're not going to say anything about it in the opening, Your Honor.

THE COURT: Well, that takes care of it then. I would like some further briefing on it or have some further argument, because all we've got was this one line. And I was out of town until Friday, and I came in and I began to think about it over the weekend. And so I'm a little -- I wanted to get some -- as long as it doesn't come up here this morning in opening, we're in good shape.

MR. LEE: We're not going to open on -- I actually think a couple of short pages may help, and then I don't think it will come up before tomorrow.

THE COURT: That's what I wanted to do tonight. Mrs. Ward didn't have anything, and she's out of town, so that will give me something to do.

Thank you, Mr. Lee.

MR. LEE: We'll keep you busy, Your Honor.

THE COURT: That's fine. Thank you.

Anything else you need guidance from?

Have I got a current exhibit list up here yet?

COURTROOM DEPUTY: Just one.

THE COURT: All right. Y'all need to get your exhibit lists.

Have y'all exchanged the exhibit list this morning?

MS. ELDERKIN: Yes, Your Honor.

MR. LEE: Yes, Your Honor.

THE COURT: Are there any disagreements? Do we have one common list, or do we have two different lists?

COURTROOM DEPUTY: I just have the Plaintiffs'.

THE COURT: Okay. I want to get them in. But there's no objection to the list each side has been furnished, is that correct, from the Plaintiff?

MS. ELDERKIN: That's correct, Your Honor.

THE COURT: All right. So those are deemed admitted. You can refer to them at anytime.

What about from the Defendants; is there any --

MR. BECK: Your Honor, there were some exhibits we talked to counsel about this morning, which our hearsay objections were sustained by Judge Everingham. They're back on the exhibit list, but I don't know whether they're going to use -- Mr. Sayles says that they're not going to be using it with the witness that he intends to put on.

So maybe the best way to do this, subject to Your Honor's approval, is to just see whether or not they're ever going to use these exhibits. And if they're not, then it's moot. If they do, then the Court can address them at that time.

THE COURT: Those that are on the list that are consistent with Judge Everingham's rulings are deemed admitted. And, you know, counsel are instructed to use those in accordance with Judge Everingham's prior rulings.

If you have something you want me to reconsider, then you will approach.

MR. BECK: Thank you.

THE COURT: We need to get another list. Yes, Mr. Sayles?

MR. SAYLES: I don't think there's an issue there. I explained it to them this morning. I

told them exactly what I'm going to use that's admitted.

THE COURT: Got the jury notebooks. What do we have in the jury notebooks over there?

LAW CLERK: You have one, Judge, there.

THE COURT: Pardon me?

LAW CLERK: You have one there.

THE COURT: Oh, I've got one.

Any objections -- just for the record, does anybody have any objections to what's included in this?

MR. BECK: We have none, Your Honor.

MS. ELDERKIN: No, Your Honor.

THE COURT: Just make one observation. This claim construction, what's here and the amount of work that went into producing this less than one page is sort of a -- what I tell everybody about trying these patent cases, I said it's claim construction that drives the train and how quick you can do that. I guess if you look at that one page, I wonder what that judge does in his spare time.

I'll see you back in here right at 8:30. We'll have a formal opening of court. I'll give some preliminary jury instructions.

And 30 minutes a side on oral argument; is that correct?

MS. ELDERKIN: Yes, Your Honor.

THE COURT: All right.

COURT SECURITY OFFICER: All rise.

(Recess.)

(Jury in.)

COURT SECURITY OFFICER: All rise.

THE COURT: All right. Please be seated.

Good morning, Ladies and Gentlemen. I have already visited with counsel, and so we are about ready to go.

I appreciate so much you being here timely. I know it's not the most convenient thing, but we've managed to dispose of some other cases that were selected in the month ahead of you, and now then we're going to take up this case.

For the record, before I give these preliminary instructions to the jury, I want to ask that the -- call on the parties for announcements and give you the opportunity and request that you, once again, introduce yourselves to the jury and those members of your team that will be participating in this case.

This is Centocor, Incorporated, and New York University versus Abbott Laboratories, Civil Action 2:07-CV-139.

What says the Plaintiffs?

MR. SAYLES: May it please the Court. Centocor Biotech, Incorporated, and New York University announce ready to proceed.

I'm Dick Sayles, counsel for the Plaintiffs. With me is Diane Elderkin, my co-counsel; Steve Maslowski, also co-counsel; Barbara Mullin, co-counsel; and at head of the table, Mr. Eric Harris, who is our client representative.

THE COURT: Thank you, Counsel.

For the Defendants?

MR. BECK: Your Honor, for the Abbott Defendants, David Beck. And with me are my colleagues, Mr. Bill Lee, Mr. Bill McElwain, Ms. Amy Wigmore, and Mr. Gil Gillam.

And may I also introduce our corporate representative, Your Honor?

THE COURT: Certainly.

MR. BECK: I would like to introduce Dr. Jochen Salfeld, who we'll be hearing from during the trial. He will be our corporate representative, Your Honor.

THE COURT: All right. Thank you.

Members of the Jury, you have previously been sworn as the jury to try this case. As the jury, you will decide the disputed questions of fact.

As the Judge, I will decide all questions of law and procedure. And from time to time during the trial and at the end of the trial, I will instruct you as to the rules of law that you must follow in making your decision.

Now, I want to -- from jury selection, I recall, according to my notes at least, that some of you have previously served on state court juries. And one of the things I learned early on in my first year and these last ten years was that jurors with that experience sometimes rely on state court procedure as to what might happen in this, the federal court.

In state court, at the end of the trial after the lawyers have argued to you, what happens is that all of the written instructions and rules of law that the Court has instructed on, you're given a copy of, to take it back to the jury room.

That will not be the case. All my instructions will be oral. I ask you to pay close attention, because now I will tell you my final instruction, if there's something you need me to repeat or something, we'll get to that. But for the most part, we will not have copies of these instructions. So I hope you will listen to them carefully.

You will recall on the date that you were

selected that you saw a film about patents. I am going to review briefly some of the things, highlights about a patent and what it is and how one is obtained.

You have in your -- in your jury notebook that's got a copy of the patent-in-suit. But this case involves a dispute relating to this particular United States patent.

Now, before summarizing the positions of the parties and the legal issues involved in the dispute, let me take a moment to explain what a patent is and how it is obtained.

The United States Constitution grants Congress the power to enact laws, quote, to promote the progress of science and useful arts by securing for limited times the authors and inventors the exclusive right to the respective writings and discoveries.

That's the end of the quote from the Constitution.

With this power, Congress enacted the patent laws. Now, patents are granted by the United States Patent & Trademark Office, referred to throughout this trial generally, as the PTO.

The process of obtaining a patent is called patent prosecution. A valid U.S. patent gives the patent owner the right, for up to 20 years from the

date that the patent application was filed, to prevent others from making, using, offering to sell, or selling the patented invention within the United States or from importing it into the United States without the patent holder's permission.

A violation of a patent owner's rights is called infringement. The patent owner may try to enforce a patent against another -- against persons believed to be infringers by a lawsuit filed in federal court.

Now, to obtain a patent, one must file an application with the PTO. The PTO is an agency of the federal government and employs trained examiners who review applications for patents.

The application includes what is called a specification, which must contain a written description of the claimed invention, telling what the invention is, how it works, how to make it, and how to use it so that others skilled in the field will know how to make and use it.

The specification concludes with one or more numbered sentences. These are the patent claims. When a patent is eventually granted by the PTO, it is the claims that define the boundaries of its protection and give the notice -- and give notice to the public of

those boundaries.

Now, after the applicant files a patent application, a PTO Patent Examiner reviews the patent application to determine whether the claims are patentable and whether the specification adequately describes the invention.

In examining a patent application, the Patent Examiner reviews records available to the PTO for what is referred to as prior art. The Examiner also will review prior art if it is submitted to the PTO by the applicant.

Prior art is defined by law, and at a later time, I will give you specific instructions as to what constitutes prior art.

However, in general, prior art includes things that existed before the claimed invention that were publicly known or used in a publicly accessible way in this country or that were patented or described in a publication in any country.

The Examiner considers, among other things, whether each claim defines an invention that is new, useful, and non-obvious in view of the prior art.

A patent lists the prior art that the Examiner considered. This list is called the cited references.

Now, after the prior art search and examination of the application, the Patent Examiner then informs the applicant in writing what the Examiner has found and whether any claim is patentable and thus will be allowed.

This writing from the Patent Examiner is called an office action. If the Examiner rejects the claims, the applicant then responds and sometimes changes the claims or submits new claims. This process, which takes place only between the Examiner and the patent applicant, may go back and forth for some time until the Examiner is satisfied that the application and claims meets the requirements for a patent.

The papers generated during this time of communicating back and forth between the Patent Examiner and the applicant make up what is called the prosecution history. All this material becomes available to the public no later than the date when the patent issues.

Now, the fact that the PTO grants a patent does not necessarily mean that any invention claimed in the patent, in fact, deserves the protection of a patent.

For example, the PTO may not have had available to it all of the information that will be presented to you. A person accused of infringement has

the right to argue here in federal court that a claimed invention in the patent is invalid because it does not meet the requirements of a patent.

Let's take a moment to look at the patents in issue. You've got -- after your glossary, you've got PX -- you've got Plaintiffs' Exhibit 1. Now, the cover page of the patent provides identifying information. That's the second page actually that we're talking about.

It has the date the patent was issued, the patent number along the top -- this long number here -- as well as the inventor's name and a filing date and a list of the cited references considered by the PTO that I mentioned to you just a moment ago.

The specification of the patent begins with this abstract, right down at the bottom of this page. And it is organized into two columns on each page. The specification ends with numbered paragraphs. You've got to go all the way over here in this particular patent -- let's see -- Column -- all the way over to Column 107.

Go right on over to next to the last two pages, and it says what is claimed is. Then it sets forth those numbered paragraphs.

Now, between the beginning abstract and

these claims are the drawings, and the drawings will illustrate various aspects of the feature of the particular invention. Then there comes this written description. They're organized into two columns, and then ending with these numbered claims.

The patent claims is what you will be focusing on not exclusively, but it's very important because it's the patent claims that determine the scope of the invention.

Now, let me talk now to you a little bit about position. That's all I'm going to say about the patent, but I wanted you to have some idea.

You will see that that has a real long number, and, generally, like it's '7 -- this one is 7,070,775. It will generally be referred to as the '775 patent, those last three numbers.

In order to help you to follow, I'm going to give you a summary of the position of the parties. And the Plaintiffs in this case are Centocor Ortho Biotech, Incorporated, referred to in this case as Centocor and New York University.

The Defendants are Abbott Laboratories, Abbott Bioresearch Center, Inc., and Abbott Biotechnology Limited. Both the parties -- I may refer to the Defendants collectively as Abbott or the

Defendants. And sometimes they will refer to the Plaintiffs collectively as the Plaintiffs.

As I -- I've given you the long number and for -- as I've told you, generally, we will all refer to this as the '775 patent.

The Plaintiffs filed suit in this Court seeking money damages from the Defendants for allegedly infringing the '775 patent by making, using, or importing, selling, and offering for sale products that the Plaintiffs argue that are covered by Claim No. 2, 3, 14, and 15 of the '775 patent.

Now, the product alleged to infringe is Abbott's product Humira. The Plaintiffs further argue that Defendants' infringement was willful.

The Defendants deny that Humira infringes any of the asserted claims of the patents at issue. In addition, the Defendants contend that the asserted claims of the patent are invalid.

Your job, ultimately, will be to decide whether Claims 2, 3, 14, and 15 of the '775 patent have been infringed and whether those claims are invalid.

Now, if you decide that any claim of any patent has been infringed and is not invalid, you will then need to decide any money damages to be awarded to the Plaintiffs to compensate them for the infringement.

You will also need to make a finding as to whether or not the infringement was willful. If you decide that any infringement was willful, that decision should not affect any damage award you give. That is for the use of the Court, and I will take willfulness into account later.

Now, it is my job as Judge to determine the meaning of any claim language that needs interpretation. You must accept the meaning I give you and use them when you decide whether any claim of the patents has been infringed and whether any claim is invalid.

You have been furnished with a copy of the meanings I have adopted on certain claim terms. That's the very last page of your notebook. It's nothing to look at at this time, but it's there when the time comes.

Now, I want to talk to you generally about the trial. Soon, the lawyers for the parties will make what is called an opening statement. Opening statements are intended to assist you in understanding the evidence. What the lawyers say is not evidence. After the opening statements, then the parties will present to you the evidence in this case.

After all of the evidence is presented,

the Court will recess for the purpose of preparing final instructions, and then the lawyers will again address you and make final arguments. And then I will instruct you on the applicable law, and then you will retire and deliberate on a verdict.

I want to say just a few words about your conduct as jurors. First, you are not to discuss this case with anyone, including your fellow jurors, members of your family, people involved in the trial, or anyone else; nor are you allowed to permit others to discuss the case with you.

If anyone approaches you and tries to talk to you about the case, please let me know about it immediately.

I want to say you've got to keep an open mind during the trial of this case. You've got to wait until both sides get to present all of their evidence, because, you know, if you make up your mind early on, it's just not fair to either party. So please follow that instruction.

And that's why I will -- that's why we instruct you not to discuss the case among yourselves until you've heard all evidence, because we don't want you deliberating among yourselves on partial evidence.

Secondly, do not read any news stories or

articles or listen to any radio or television reports about the case or any -- about anyone who has anything to do with it. There may be something in the newspapers; there may not. It may be on some of the local news channels, or it may not.

Another important thing is do not do any research such as consulting dictionaries, searching the internet, or using any other reference materials. And do not make any investigations about the case on your own.

Now, there have been recent, tragedies in a sense, for a trial judge, not that he was hurt, but eight weeks in trial, it turns out the lawyers were communicating with somebody outside in one case on their cell phones about what some terms meant or didn't mean. And then that's happened at another time. So please do not do anything like that.

Just remember that instruction: Don't talk to anyone about this case, and don't do any research or try to learn anything.

Now, if you need to communicate something with me, you need to just give a copy to the court security officer in writing of what you want me to know about, and I'll decide if there's anything I can do or we will act on it.

Finally, do not -- this is what I've already said to you once, but I want to emphasize it. Do not make up your mind about what the verdict should be until you have gone to the jury room to decide the case, and you and your fellow jurors have discussed the evidence. Keep an open mind.

Now, during this trial, as in all trials, it will probably be necessary that I consult with the lawyers outside your hearing to conduct a part of the trial outside your presence. I will handle these matters as briefly and as conveniently for you as I can, but you should remember that this is a necessary part of any trial.

With respect to evidence, I want to tell you, the evidence you are to consider in deciding what the facts are consist of the sworn testimony of any witness, the exhibits which are to be received into evidence.

Let me comment briefly to say that the lawyers have worked extremely hard with myself and Magistrate Judge Everingham to minimize the amount of time talking about admissibility of exhibits. We've had numerous hearings, and so there won't be a lot of wasted time. These exhibits have been previously ruled on.

So they are to be complimented, both

sides, because it's going to make your job -- number one, it's going to save you a lot of time, and it will make your job easier.

Also, you are to consider any stipulations that the parties agree to. If the parties stipulate something as to a fact, you are to consider that fact conclusively proved.

Now, what is not evidence?

The following things are not evidence, and you must not consider them as evidence in deciding the facts of this case. I've already said statements and arguments of the attorneys; questions and objections of the attorneys; testimony that I instruct you to disregard, and anything that you may see or hear when the Court is not in session, even if what you see or hear is done or said by one of the parties or by one of the witnesses.

Now, evidence, the type of evidence may be direct or circumstantial.

Now, direct evidence is direct proof of a fact, such as the testimony of an eyewitness about what that witness personally saw or heard or did.

Circumstantial evidence is proof of one or more facts from which you find another fact.

You should consider both kinds of

evidence. The law makes no distinction between the weight to be given either direct or circumstantial evidence. It is for you to decide how much weight to give any evidence.

In deciding the facts in this case, you may have to decide which testimony to believe and which testimony not to believe. You may believe everything a witness says, part of it, or none at all.

In considering the testimony of any witness, you may take into account the opportunity and the ability of the witness to see, hear, or know the things testified to, the witness' memory, the witness' manner while testifying, the witness' interest in the outcome of the case, and any bias or prejudice; whether other evidence contradicted the witness' testimony; the reasonableness of the witness' testimony in light of all the evidence; and any other factors that bear on believability.

The weight of the evidence as to a fact does not necessarily depend upon the number of witnesses who testify. Remember, you are the exclusive judges of the facts.

Now, you must consider only the evidence in this case; however, you may draw such reasonable inferences from the testimony and exhibits as you feel

are justified in light of common experience. You may make deductions and reach conclusions that reason and common sense lead you to make from the testimony and evidence.

With respect to common sense, folks, it is your collective wisdom and your collective common sense that separates you from everyone else in this trial. Do not leave your common sense outside the courtroom. Those are the best tools you have to resolve the fact questions that you must.

The testimony of a single witness may be sufficient to prove any fact, even if a greater number of witnesses that may have testified to the contrary if, after considering all the other evidence, you believe the single witness.

Now, when a party has the burden of proof on any claim or affirmative defense by a preponderance of the evidence, it means that you must be persuaded by the evidence that the claim or affirmative defense is more likely true than not true. You should base your decision on all the evidence, regardless of which party presented it.

I believe I gave you this example. We've got the scales of justice here. We're going to start this trial, we start off even. We've heard no evidence.

At the close of the evidence, after hearing my final instructions on preponderance of the evidence, if you believe that the party who has the burden of proof by a preponderance of the evidence is something more likely true than not, that means the scales are tipped ever so slightly in favor of that party. That's the preponderance of the evidence standard.

Now, you have another burden that you're going to have to consider -- burden of proof. That's the clear and convincing evidence standard.

Now, when a party has the burden of proving any claim or defense by clear and convincing evidence, it means that the party must persuade you that it is highly probable that the facts are as that party contends. Such evidence requires a higher standard of proof than by a preponderance of the evidence.

Again, you should base your decision on all the evidence, regardless of which party presented it.

Thinking of the scales of justice again rather than tipping ever so slightly, they've got to tip more like something like this (indicates) for the clear and convincing standard.

That is not to be confused nor will you be required to apply the burden of proof we hear a lot

about on TV. That's beyond a reasonable doubt in a criminal case. The scales have to tip all the way for someone to meet that burden of proof.

Do not confuse the clear and convincing standard with that. It is a lesser burden than beyond a reasonable doubt.

Now, you're going to hear in this case from a number of expert witnesses. I want to talk to you.

When the knowledge of technical subject matter may be helpful to the jury, a person who has special training or experience in that technical field, called an expert witness, is permitted to state his or her opinion on those technical matters. However, you are not required to accept that opinion.

As with any other witness, it is up to you, as exclusive judges of the facts, to decide whether or not to rely on it and how much weight to give the testimony.

Now, during this trial, certain testimony will be presented to you by way of deposition.

Anything other than video depositions?

MR. SAYLES: May it please the Court. There's one deposition to be read in, and there's some requests for admissions that will come up.

THE COURT: Well, we'll deal with the requests for admissions separately.

Nearly all the deposition testimony is done by video, so you will get to see the witness testify and hear the testimony. But deposition is testimony of a witness who for some reason cannot be present to testify from the witness stand is usually presented either in writing or by way of video under oath. One that will be read to you, the testimony will be under oath.

Now, such testimony, whether it be read to you out of a deposition or placed on the screen through a video, when you hear the witness testify, such testimony is entitled to the same consideration, and, insofar as possible, is to be judged as to credibility, weight, and otherwise considered by the jury in the same way as if the witness had been present and had given from the witness stand the testimony that is shown on the screen or that is read to you from the deposition.

Now, we've got the lawyers in this case that have worked extremely hard, and they're advocates for their clients. A lawyer is ethically and legally obligated to zealously assert his or her client's position under the rules of our adversary system. And by presenting the best case possible on behalf of

their clients, the lawyers, hopefully, will enable you, the jurors, to weigh the evidence and determine the truth, and arrive at a just verdict based on the evidence.

This adversary system of justice has served us well for over 200 years. And trial lawyers have then and continue to be a critical part of the process. And in performing their duties, it is the duty of the attorney on each side of this case to object when the other side offers testimony or other evidence which the attorney believes is not properly admissible.

Now, upon allowing testimony or other evidence to be introduced over the objection of the attorney, the Court does not, unless expressly stated, indicate any opinion as to the weight or the effect of such evidence.

As I've told you before, you, the jurors, are the sole judges of the credibility of all the witnesses and the weight and effect of all evidence. However, when the Court sustains an objection to a question addressed to the witness, the jury must disregard the question entirely and may draw no inferences from the wording of it or speculate as to what the witness would have said, if permitted to answer the question.

Now, the law of the United States permits the judge to comment to the jury on the evidence in a case, but such comments are only expressions of the judge's opinions as to facts. And the jury may disregard them entirely, since the jurors are the sole judges of the facts.

All right. Is the Rule to be invoked in this case?

MR. BECK: It is, Your Honor. We wish to do so.

THE COURT: All right.

MR. SAYLES: We have agreed that experts are excused from the implementation of the Rule.

THE COURT: Well, that's the Court's practice.

Are there witnesses in the courtroom at this time that will be subject to the Rule?

MR. SAYLES: Yes, sir, there are.

THE COURT: All right. If you'll have them come forward and just stand right there, because I want to give them some instructions.

MR. BECK: Your Honor, do you wish experts, also?

THE COURT: No, not experts. Non-experts, fact witnesses, non-experts.

MR. SAYLES: Inside the rail, Your Honor?

THE COURT: No, they're fine. I just want to make sure they can hear me.

The Rule has been invoked in this case, and it applies to -- I've excused the expert witnesses, but those who are non-expert witnesses, fact witnesses, what that means is that from this point forward, you are under the Rule.

And that means you cannot discuss your testimony with anyone, other than the lawyers in this case. And when you're discussing your testimony with the lawyers, you have the duty the same as the lawyer does, to see to it that you are outside of earshot of any other person so that they cannot hear these discussions. So that is your duty as well as the lawyers'.

I'm sure there will be witnesses, maybe perhaps other than these, but it's the duty of the lawyer to make sure that any other witness not present here at this time is aware that the Rule has been invoked and what the Court's instructions are.

You may be seated. You may remain in the courtroom until opening statements are completed. You may hear the opening statements, but then you will be required to place yourself outside the courtroom.

Thank you very much. You may be seated.

All right. At this time, we'll hear from the attorneys for the Plaintiffs.

I will give you a five-minute warning.

MS. ELDERKIN: I was going to ask for that. Thank you very much.

May it please the Court, Counsel, Ladies and Gentlemen of the Jury.

If someone uses someone else's property without permission, they should pay for it.

My name is Dianne Elderkin, and along with counsel at table who were introduced previously, I represent the Plaintiffs in this case, Centocor Ortho Biotech and New York University.

This trial is about drugs, revolutionary drugs, drugs that have changed the way that very devastating diseases are treated, diseases such as rheumatoid arthritis and Crohn's disease.

You're going to hear that because the U.S. Patent & Trademark Office, an agency of the U.S. government, found that those drugs were new and useful, it issued this patent, the '775 patent, that is in your binders.

It was issued on Independence Day, July 4th, 2006.

You heard that when the government issues a patent, a valid patent under discovery, that means that no one else can use the invention in that patent that's claimed in that patent without permission for a set period of years.

We're going to show you that Abbott chose to disregard those rules. They knowingly used this patent, the invention, when they sold their drug, Humira, without permission from Centocor.

This trial is about Centocor's claim for damages for Abbott's use of our patented invention.

Now, you may not recognize the name Centocor, but you probably recognize the name Johnson & Johnson. Johnson & Johnson companies make all kinds of products like Band-Aids and Tylenol -- Tylenol, Band-Aids, baby oil, baby powders.

But there are also Johnson & Johnson companies that make prescription drugs, drugs that treat things like cancer, infections, pain, and even Alzheimer's disease.

Well, Centocor is a Johnson & Johnson company, and it's a Johnson & Johnson company that makes a special kind of drug called antibodies for treating diseases. And the patent in this trial, the '775 patent, was granted to scientists at Centocor and at

NYU, New York University, because they invented a special kind of antibody that can be used to treat diseases such as rheumatoid arthritis and Crohn's disease.

You are going to hear that rheumatoid arthritis is different from the kind of arthritis, the more common arthritis that we often hear about where your joints just sort of wear out. Rheumatoid arthritis is really much worse than that.

It's a disease where inflammation attacks the joints in your body and can actually cause them to deform. So sometimes patients who have rheumatoid arthritis can't accomplish the simplest of tasks or even get out of bed. And it affects over a million people in the United States.

Another disease you're going to hear about is Crohn's disease. That's another terrible disease, and it's a inflammation disease that affects your intestines, your bowel, and can have devastating and debilitating consequences, including sometimes the need for repeated surgery to remove the intestine.

Now, rheumatoid arthritis and Crohn's disease probably seem very different to you. One affects joints; one affects the bowels; but they really have something in common. They're both caused by

inflammation, and they're both caused by an inflammation because our bodies overproduce a particular protein called tumor necrosis factor. You're going to hear a lot about that.

Tumor necrosis factor alpha, which is abbreviated as TNF. The TNF is actually a good protein; we all have it in our bodies, and it's a necessary part of our immune system. But sometimes the body can overproduce it, and when it's overproduced, it can cause the kinds of inflammation that leads to these horrible diseases.

Now, in the 1980s and 1990s, there really were not adequate treatments for diseases like rheumatoid arthritis and Crohn's disease for a lot of patients. There simply was nothing that really worked for everybody.

The inventors on the Centocor and NYU patent theorized that one way to treat the diseases would be to go into the body somehow and capture this excess TNF that your body makes and somehow remove it from your body.

Now, at the time, that probably seemed like science fiction, but that's exactly what Centocor's and NYU's scientists did. They invented and made antibodies that were able to get -- that when

administered into your body were able to find the TNF in your blood, hook onto it, and then have it removed from your body so that it couldn't cause the inflammation that was causing problems.

And, in fact, they discovered and were awarded this patent on two types of antibodies for doing that. One's called chimeric and one is called human. So what are antibodies?

Well, antibodies are very different from the kinds of drugs that we're mostly used to, the kind of drugs that a pharmacist might be able to mix up behind the counter at the drugstore.

Antibodies are proteins, and they are made in living organisms such as in our bodies. Our bodies make antibodies to protect us against germs, things like bacteria and viruses.

So, for example, if you had the chicken pox, what happens is your body sees that foreign chicken pox virus and makes antibodies to it, and eventually those antibodies remove the virus and take it out of your body and you get better.

Also, those antibodies then stay in your body. So if you're ever exposed to the chicken pox virus again, those antibodies are there to go find the virus and remove it from your body, and you're better

possibly before you even knew that you had the virus.

Centocor was one of the first companies to develop man-made antibodies to treat diseases, and the particular antibodies that Centocor and New York University invented are antibodies that are specific to, that bind to that TNF protein that's in our bodies. One of the inventors of the patent, Dr. John Ghrayeb will be here to tell you about the invention that is described in the patent and the work that went into it.

The work to develop these antibodies began in the late 1980s. Long before Centocor was a Johnson & Johnson company, it was a pretty small startup company at the time. It didn't have a lot of money, but it had some great inventors and great ideas.

And so it needed some help, so it entered into an agreement with New York University, NYU, to do some research together. And what happened is the New York University scientists were charged with looking for antibodies from mice that might bind to TNF.

What they did, rather ingeniously, is the NYU inventors took mice and they injected them with human TNF-alpha, the kind of TNF-alpha that we have in our bodies.

Because that's a human protein and not a mouse protein, it was foreign to the mice, and so the

mice produced antibodies to the TNF. They did that and the New York -- the NYU scientists were able to isolate those antibodies that the mice made. One of them was a very special antibody, which is called A2. You'll hear a lot about that during the trial.

But finding A2 was really just the beginning of the story. Finding A2 was sort of like finding a needle in a haystack, because the mice -- the mouse made many, many more antibodies than that. So they had to first find the A2 antibody and determine that it was the special one, that it was capable of going in and binding to TNF tightly and holding onto it, and also that it was capable basically of putting the TNF out of business. Neutralizing it is the term you'll hear.

When the antibody bound to TNF, it was able to keep the TNF from doing the bad things, causing the inflammation that causes problems in the body.

Now since this A2 antibody that the NYU scientists found was entirely made in a mouse and from mouse cells, it wouldn't work very well for long-term treatment in humans.

So then the Centocor scientists got involved, and what they did is they took the A2 antibody and made it work better as a drug for humans.

What they did is they took a large part of the A2 antibody -- because these are very large molecules -- they took a large part of it, and they replaced it with a part of an antibody from a human. The resulting antibody was called cA2. The C stands for the term chimeric. Chimeric is a term that means that it's an antibody that has pieces from two different sources. In the case of cA2, the two sources are mouse and human.

So how did they do that; how did they make an antibody that was part mouse and part human?

Well, you'll hear that all antibodies have something in common. Whether it's a mouse antibody or a human antibody, all antibodies are made from the same building blocks, 20 building blocks. They are called amino acids, and they are the same amino acids if it's a mouse antibody or a human antibody or a goat antibody or a rabbit antibody. They are all the same. The difference is only how those building blocks are put together.

So how does one determine how the building blocks are put together?

The instructions are in DNA. You've probably all heard about DNA. It's the genetic material that's in every cell in our bodies. And DNA is like the

instruction booklet. It has the instructions for how the 20 different amino acid building blocks should be put together to make any protein but including an antibody.

So what the Centocor inventors did is they took some of the DNA for making the mouse antibody, an instruction booklet for making mouse antibodies, and they combined that with some DNA from some human DNA, an instruction book for making human DNA. They combined it into a new piece of DNA, a new instruction book for making a new man-made antibody. And that's what they did. That's how they made the cA2.

And Dr. Ghrayeb, one of the inventors, will tell you how that was done, to make a new antibody. The technology for doing this has a name. It's called recombinant DNA technology. That's because you combine two pieces of antibodies -- two pieces of DNA -- I'm sorry -- to make a new piece of DNA, recombinant DNA.

Now, the Centocor/NYU inventors further discovered that they could make another type of TNF antibody that had the great characteristics of A2, that initial mouse antibody that binds tightly and removes the bad TNF from the system.

They thought that they could make a new antibody, not necessarily a chimeric one, as the one I

just showed you, but they could also make a human antibody.

The antibodies that are made this way that we talk about as human antibodies are really not existing naturally in our bodies at all. We call them human because they're both -- they're made solely from human DNA, human DNA instruction booklets. But they don't exist anywhere naturally in our bodies.

And the inventors disclosed that they could make human antibodies, according to their invention, in an application that they filed in February of 1994. That will be an important date to remember, February of 1994.

And you will see that right in their patent in the summary of the invention section, which is in Column 5 of the patent, they talk about how the anti-TNF antibodies of their invention are intended to include both chimeric antibodies; that's one where, for example, part is mouse and part is human; and also human antibodies. And they added that reference to human antibodies in February of 1994.

Now, the process of making an antibody into a drug, once you have this great antibody in the lab -- but the process of making it into a drug, testing it on animals and humans and getting governmental

approval from the FDA takes a long time and costs tens of millions of dollars.

Centocor was still small. They didn't have the money to explore both types of antibodies, chimeric antibodies and human antibodies, and bring both to market right away. And they knew that their chimeric antibody, cA2, was a really great antibody and showed great potential. They wanted to get that out into the marketplace so that it could help patients right away.

So cA2 is the first antibody that they pursued in clinical trials, testing with patients. And you're going to hear about some of the early tests, especially with cA2.

Some doctors in Europe tested cA2 in patients, patients who had rheumatoid arthritis and Crohn's Disease, and the results were dramatic, remarkable.

For example, you're going to hear about one patient, a 16-year-old girl in Holland, who had severe Crohn's Disease. She was very, very sick, and her doctor thought it was the point where he was going to have to do an operation to remove her entire colon. You can imagine how devastating that is, especially for a 16-year-old girl.

The doctor heard about the work that was

being done with cA2 and rheumatoid arthritis, and even though cA2 hadn't been approved yet by any governmental agency for use in patients, he asked for a sample of it on a compassionate use basis to give his patient.

He got it. It was administered to the girl, and you'll hear that within a week, after one infusion of cA2, she felt better, she was gaining weight, and they didn't need to do surgery on her. The cA2 worked in patients.

Now, in 1998, after many more years with much larger tests in patients, Centocor received approval from the Food & Drug Administration, the U.S. governmental agency, to sell its product, cA2, which is called Remicade, for Crohn's Disease.

A year -- about a year later, they got approval to sell it for rheumatoid arthritis. And as you can see on the slide, you'll hear that there were other approvals in subsequent years for other diseases, and ankyloses, spondylitis, psoriatic arthritis, and psoriasis.

For 10 years now since Remicade was first introduced in 1999, it has safely and effectively treated tens of thousands of patients who have these horrible diseases.

Now, the remarkable results that Centocor

was getting with cA2 didn't go unnoticed. Johnson & Johnson, as we know, a big company with a big pharmaceutical business, was very impressed with the results of its hearing about the tests of cA2 in patients.

And in 1999, it came in and acquired Centocor. It paid $5 billion for Centocor. At that time, that was the largest acquisition Johnson & Johnson had ever made.

And in 2000, Remicade was awarded a very prestigious prize. It's called the Galien Prize or the Prix Galien. It's like the Nobel Prize, the equivalent in the pharmaceutical industry, and it awards pharmaceutical research and innovation.

Now, it turns out that Abbott, another large pharmaceutical company, also wanted in on the anti-TNF market anti-TNF antibody market.

So in 1999, after some of the great results of the clinical testing of Centocor's cA2 antibody had been made public, executives from Abbott actually talked to Centocor about making an offer to buy Centocor, but they weren't willing to pay as much for Centocor as Johnson & Johnson was, so those talks went no further.

But Abbott didn't give up on acquiring

its own anti-TNF antibody. In 2001, Noel, which was the pharmaceutical arm of a giant German company, BASF, Noel had a TNF antibody which it was developing.

Abbott bought Noel, and then beginning in 2003 started selling that antibody that Noel had started to develop. That antibody is called Humira, and that's the product that's accused of infringement in this case.

Now, Abbott followed in Centocor's footsteps. Just as Remicade was introduced in 1999 and got approvals for all these diseases, Humira, Abbott's product, was introduced in 2003, and it got approval for all the same diseases after Centocor had gotten those approvals first.

The problem is -- and Humira is a great product, and we don't deny it. It has been a wonderful product both for Abbott and for patients. It is an excellent, excellent product that has helped many people, just as Remicade has. The problem is, though, it infringes Centocor's patent.

The next slide summarizes the claims that are in issue in this case. And as Judge Ward explained, the claims at the end of the patent, those numbered paragraphs, are what define what's protected by this patent, and we're going to show you that Humira meets every single one of the requirements of the claims.

In fact, for all the red checkmarks there, Abbott doesn't even dispute that those requirements are met by Humira. The only dispute is over the blue checkmarks, which is the requirement about competitive inhibition, and you'll hear a lot about that.

But you're going to hear from some witnesses of Centocor. You're going to hear from Susan Tam, who is a Centocor scientist who did testing on Humira with respect to that claim element.

And then you're going to hear from Dr. Greg Adams, who's an expert in this field, who took Susan Tam's test results, analyzed them, and concluded that Humira does meet that claim element. And he'll be here to explain that to you.

And I want you to listen real carefully as to whether you hear anything from Abbott, if they present you any tests to rebut what Ms. Tam did and what Dr. Adams will testify about.

Now, I want to explain one more thing to you about the Centocor and the NYU patent. I told you the scientists worked on the antibodies that came -- or led to this patent in the 1990s and that they filed their first patent application in 1991.

The patent issued in 2006, but very

importantly, you'll hear that February 1994 is an important date for our patent. That's because the application which was filed with the Patent Office in February of 1994 expressly discloses human antibodies and how to make them.

You'll also hear that it's not uncommon that a patent can issue many years after the first patent application. So even though the first patent application was filed back in 1991, it's not uncommon that a patent might not issue, as this one, until 2006. So this patent did issue in July 2006, but Abbott knew it was coming at least six months earlier. The Patent Office told Centocor back in December 2005, six months before the patent issued, that it was going to allow the patent.

You're going to hear from Ken Dow, Centocor's patent lawyer, that when Centocor heard from the Patent Office that it was going to allow the claims in this patent, Centocor went to Abbott and told them, we're going to get a patent. You should look at these claims that the Patent Office is going to allow, and you can look at the claims on the -- on the website that's available to the public.

You're also going to hear from Joseph Scodari, who was a business executive at Centocor and

Johnson & Johnson at the time. You're going to hear him say that in early 2006, he told his counterparts at Abbott that Humira was going to infringe this patent. Centocor offered to let Abbott use the '775 patent. That's called a license. But Abbott refused to pay a fair amount for that permission, so it didn't get a license.

Instead, Abbott continued to sell Humira, and we allege they continue to infringe this patent knowingly and willfully, and they haven't paid Centocor or NYU a dime for doing so.

And that's why we're here. We're asking you to award Centocor fair compensation for Abbott's use of our invention, of our property. Centocor, as I said, has been an enormously successful product for Abbott, and it's been great for patients.

In fact, Humira also got the same prize that Remicade had earlier gotten, the Galien Prize, showing what a great product it is. And that's not surprising, because as we'll show you, Humira is also part of the same groundbreaking invention that is claimed and disclosed in this patent.

And Abbott even got some patents of its own on Humira, but you're not going to hear a single witness, you're not going to hear a single witness say

that because Abbott got its own patents, that it can't also be infringing our patent. That's an important thing to remember.

Now, I'd like to tell you a little bit about the damages that Centocor is seeking.

Since this patent issued in July 2006, Abbott has sold over $11 billion worth of Humira, and we're asking you to award damages for approximately 2.1 billion to Centocor.

That's a big number, folks. But we're going to show you that even if you were to award Centocor the $2.1 billion we're asking for, Abbott would still be left with more than that in profits.

You're going to hear from Rob Bazemore, one of Centocor's marketing executives, about how Humira and Remicade compete with one another in the marketplace.

And you'll hear from Dr. Richard Gering, a very experienced economist, that because Abbott has been selling Humira, Centocor has lost some sales that it would have made otherwise.

If Humira hadn't been on the market infringing our patents, as we contend, Remicade would have made more sales. And we are asking you to award us the profits we would have made had Abbott not been on

the market with its infringing Humira.

Dr. Gering is also going to explain to you that Centocor should get a royalty from Abbott on some of its sales of Humira.

In other words, a certain portion of the money that Abbott has gotten for selling Humira should come to Centocor. Those are the two numbers that add up to the $2.1 billion that we'll ask you for.

Now, Abbott has its own damages expert, and we expect that he's going to disagree with these numbers. Dr. Gering -- he'll disagree with Dr. Gering. We expect that he'll say that the drugs Remicade and Humira, they don't really compete with one another. Remicade doesn't lose any sales because Humira was on the market, and therefore, he'll say that Abbott only owes Centocor $250 million.

But you'll get to weigh the evidence on that. You'll get to weigh the evidence about whether there really is competition and whether Remicade has lost sales to Humira in view of documents like this one, Plaintiff's Exhibit 106.

This is an Abbott marketing document talking about Humira where it says, Take share from Remicade. Well, that wasn't a document created for litigation, and you'll get to see those kinds of

documents and weigh the evidence.

Changing gears a little bit, I told you that Abbott does not have permission from Centocor to sell Remicade. Well, there's a slight exception to that.

You're going to hear that last year, Abbott asked an arbitrator to decide whether Abbott had permission to sell Humira under our patent.

THE COURT: Five minutes.

MS. ELDERKIN: Thank you.

What happened is the arbitrator ruled that Abbott does not --

MR. LEE: I object, Your Honor. May we approach?

THE COURT: Yes.

(Bench conference.)

MR. LEE: I understood that the rules were that we could say that Abbott had a license, but we weren't referring to any of the other proceedings or what the arbitrators did because of the confidentiality.

MS. ELDERKIN: There's agreed upon stipulation that there is no license for sales that are not co-administered and the arbitration award itself is an admitted exhibit.

MR. LEE: Right. Right. But just the

award. We're not talking about what the -- I don't mind them per se saying that there was an arbitration and we have a license, but that's -- I understood that's as far as we could go.

MS. ELDERKIN: The award expressly says that Abbott is not licensed for sales that are not co-administered, but they are licensed for sales that are administered.

MR. LEE: That's the problem. He didn't find they were not licensed; he just found that under the facts in that proceeding, we were licensed for co-administration.

But there's no finding that we were not licensed otherwise except under that license agreement that was involved there. That's the problem.

THE COURT: I thought the award -- she just -- what's correct about whether the award is an exhibit?

MR. LEE: Yeah.

MS. ELDERKIN: The award is an exhibit.

MR. LEE: What -- if she wants to say it was in the reward, that's fine, Your Honor.

THE COURT: Well, let's stay right now with what's in the award.

MR. LEE: Right.

MS. ELDERKIN: Yes, sir.

(Bench conference concluded.)

MS. ELDERKIN: Does that go against my time, Your Honor?

THE COURT: No. I stopped the clock.

MS. ELDERKIN: Thank you.

What you're going to hear, you're going to see the award that the arbitrator made, and what he said is that only a portion of Abbott's sales are licensed.

And we're not asking for damages for that portion of Abbott's sales of Humira -- for Humira. That's only a fraction of their total sales. You're going to hear that Dr. Gering took that out of the pool when he was determining what those damages should be.

You're going to hear that Centocor has no problem competing with Abbott in the marketplace. Centocor believes that it's better for patients to have a number of good effective drugs for treating these bad diseases.

In fact, in recent years, Centocor developed another anti-TNF antibody that will compete with both Remicade and Humira. Just this past April, Centocor introduced this new product. It's called Simponi, and it is an anti-TNF antibody. It's a human

anti-TNF antibody.

And Centocor brought that to the marketplace to reach those patients who might prefer a drug that can be self-administered by a shot rather than an IV infusion or for whom the other drugs, such as Remicade and Humira, don't work.

You'll also hear that before selling Simponi, Centocor went to Abbott and got a license to use some of Abbott's patents that it might possibly need for selling Simponi. It went and got permission, because that's the way it's supposed to be done.

Centocor's not seeking to take Humira off the market. All we want is to compete fair and square. But it's not fair and square competition when Abbott doesn't pay to use our property. We're asking for fair compensation for Abbott's use of our property, our patent.

And finally, we're going to ask you to determine that Abbott's infringement was knowingly -- was knowingly willful.

I don't know what Abbott's going to say about this, but you're probably going to hear some excuses for their conduct. They might deny that they infringe, but, again, watch for whether they show you a single test where they tested Humira to rebut the

testing that we did.

And then they say that the patent is invalid. They may try to prove by the clear-and-convincing standard that the Patent Office made a mistake, but you'll get too weigh all the evidence on that at the end.

Bottom line, if someone uses someone else's property, they should pay for it.

This is an important case. And we know it's a real imposition for you to be away from your busy lives for a week. We really thank you for your participation, and we look forward to presenting the evidence to you.

THE COURT: Thank you, Ms. Elderkin. Mr. Lee.

MR. LEE: Thank you, Your Honor. Just a moment for the electronics.

(Pause in proceedings.)

THE COURT: Proceed.

MR. LEE: If it please the Court.

Good morning, Ladies and Gentlemen. My name is Bill Lee, and together with my colleague, David Beck, Amy Wigmore, Gil Gillam, and Bill McElwain, I represent Abbott.

As you now know, Centocor's accusing

Abbott of patent infringement, patent infringement based upon Abbott's selling of its groundbreaking drug, Humira, and it's asking you for literally billions of dollars.

And if Ms. Elderkin's opening -- not evidence nor is mine -- if her opening were an accurate statement of all the facts, you might be asking yourselves, well, why are we here?

Well, we're here, because there is another side to the story, precisely the reason that His Honor asked you to keep an open mind. And this opening is my opportunity, not to present you with evidence, but to tell you Abbott's side of the story.

The evidence that we will provide you will fill in many of the holes in the story Centocor has offered you, and that evidence will demonstrate to you that Centocor is asking you to give it billions of dollars for a very important product that Abbott was first to make, the first to bring to patients, the first to get Food & Drug Administration approval, the first to bring to doctors and the medical community.

That evidence will demonstrate that Centocor cannot succeed in this case for two independent reasons.

First, it will not be able to comply with

the requirements that His Honor described to you for a valid patent. It will not be able to demonstrate that its patent satisfies those requirements.

And second, it will not be able to demonstrate to you that it can carry its burden of demonstrating that our infringement was -- that we infringed or that any infringement was willful.

Let me begin by introducing you to our client, to our company. Abbott is one of the leading healthcare companies in the world. It is an innovator. It is a creator. It is an inventor.

It has developed drugs to treat diseases, such as heart disease, cancer, AIDS, tools to treat folks with diabetes. It makes surgical devices. It makes test equipment. It makes Similac infant formula.

Millions upon millions of people have benefited from Abbott's inventions. Just as Ms. Elderkin had a story of someone who was successfully treated with Remicade, their product, there are millions of stories about Abbott.

And Abbott invests literally billions of dollars every year in research and development to develop those products and bring them to patients and to doctors and to the medical community.

Now, Humira is one of those life-altering

products that Abbott has brought to patients. Also is a first-of-its-kind product, and this is something that Centocor didn't tell you in its opening. Centocor didn't tell you that Humira was the first fully human antibody therapy for a number of diseases, such as rheumatoid arthritis and Crohn's disease.

In fact, it was the first fully human antibody of any kind, any kind ever on the United States market.

Now, as Ms. Elderkin said, these diseases are serious and painful diseases. They can result in joint deformity, loss of function, chronic pain, and complications in our digestive systems.

What makes Humira a groundbreaking drug is the fact that it is high affinity, meaning that it sticks to the target; neutralizing, meaning it works; and it's fully human. There are no mouse parts; there are no rabbit parts; there are no rodent parts, as the patent, the '775 patent, describes.

Now, to understand the importance of Abbott's fully human antibody, it will be important to understand the development of antibody treatments which are used to treat what are called autoimmune disorders.

In autoimmune disorders, our bodies, which have a defense system, the defense system starts

to attack itself and attack our healthy cells. Not what the body is supposed to do.

So for folks who have rheumatoid arthritis or Crohn's Disease, our bodies are not functioning correctly, and the immune system is attacking healthy cells.

In rheumatoid arthritis, for instance, this TNF-alpha that Ms. Elderkin mentioned to you, a protein that is part of our system becomes overly active, and it starts attacking our joints, just as is shown on the x-rays.

Scientists recognized that one way to treat these diseases were with antibodies. If you have something like TNF-alpha that is attacking our joints, what do you do? And scientists recognized that you could use what are called antibodies.

Antibodies are also part of our immune system. And what they are, are things, actual things, that attach themselves to the bad things, to the virus, to the bacteria, and then escort them out of our body or make them not harmful.

Scientists thought that there was -- could be an antibody to TNF-alpha. If we could develop an antibody to TNF-alpha that doctors could inject into patients, then we could address the problem created by

our own bodies attacking themselves.

Now, what the evidence will show you is, as scientists approached this problem, the problem of finding antibodies, there were different types of antibodies that they explored. And the different types of antibodies become critical to the issues before you. When scientists began, they started by making mouse antibodies. They are antibodies completely made from mouse parts. These antibodies were made, as Ms. Elderkin described, by injecting mice with something like TNF and extracting the antibodies.

But the effect of this -- of these mouse antibodies was limited. It was limited because they're mouse parts.

And when you put a mouse part into our bodies, our body say, Wait a minute; this is a foreign substance; I don't want this foreign substance in me; I'm going to generate antibodies to remove it from my body, just like you would if you had a virus, just like you would if you had the flu.

What does it mean? It means that if you had a mouse antibody, if you really had discovered a mouse antibody that could attach itself to the TNF, it couldn't work for very long, because pretty soon our bodies would say, I know that doesn't belong in my body;

I know I want it out of here; and it would be ineffective.

But equally important, because it came from mouse or rabbit or rodent parts, it created the risk of medical complications, which you've heard referred to as side effects, unwanted effects that come from taking the product.

So what happened? This led to -- this led scientists to develop new laboratory techniques to combine genes from two different species, from mouse and human, some portion from the human, some portion from the mouse. These are called chimeric antibodies. And this is what Centocor made. A chimeric antibody comes from the Greek word chimera, which refers the combination of a lion, a snake, and a goat. But the patent itself tells us that a chimeric antibody has something that's part human but also part mouse, rabbit, rat, or hamster.

Now, these antibodies were better. They were better because they were part human and part mouse. But they still triggered unwanted responses by our bodies.

Our bodies still recognized that there was part of them that were not human, and as a consequence, said, we don't want this here; we need to

get it out of our bodies, and generate an immune response.

So what did scientists do? They went further and said, well, then what we need is something that is fully human. We need something that when you put it in our bodies, our bodies won't say it's foreign. They won't reject it. They won't have side effects.

And what the evidence is going to demonstrate to you is, on this critical step forward, this critical step of creativity, it was Abbott, not Centocor, that made the first fully human antibody. And that first fully human antibody was Humira.

Humira was completely developed -- developed independently by Abbott. You'll hear no evidence that Abbott somehow had the chimeric antibody of Centocor and used it to develop Humira.

Humira was the first, the very first fully human antibody, to treat diseases like rheumatoid arthritis in the world until one month ago when Centocor brought to market its first fully human antibody.

Now, Humira is, as Ms. Elderkin said, an award-winning medical treatment. It has treated thousands upon thousands of patients. It received the Galien Prize in 2007, the Nobel Prize in pharmaceuticals.

And I'm going to ask Mr. Beck to hold up the prize, which is right behind him.

This is the Galien Prize.

Now, the irony or the interesting fact here is that when Abbott was awarded the Galien Prize, the Nobel Prize for pharmaceuticals, guess who was on the committee that gave us the prize? Their inventor. The inventor of the patent they now claim is infringed voted to give us the pharmaceutical Nobel Prize.

Now, you may be asking yourselves, now, if Abbott was first to make, first to patent, first to bring to patients, why are we here?

Well, we're here because Centocor is saying, well, we have a patent that covers Humira.

Now, Ms. Elderkin has told you that we are challenging the validity of the four claims that are before you. We are.

And you may ask yourselves, if the Patent Office already decided that Centocor is entitled to a patent, what is our role?

The answer is that in our legal system, the patent laws specifically make you part of the process just for the reasons that His Honor told you. The reason you are so important is that the process to get a patent is a secret one. Only Centocor and the

Patent Office participated.

If Abbott had dialed up and said, could you tell us what's going on down there, we would have been told, none of your business. Abbott could not and did not participate.

Now -- now, our Patent Office is a fine Patent Office, but as the video and Your Honor's instructions have demonstrated, it can only make decisions based upon the information that it has before it.

We are going to present to you evidence that the Patent Office did not have and could not consider, and that is the reason that we have an opportunity to present our case, our side of the story to you.

And as His Honor explained this morning, it will be for you to decide those issues with a full deck of cards for the first time.

Now, the key to this case will be the chronology of events. And I'm going to take some of the things that Centocor mentioned to you and put them in order. Because at the end of the evidence on Thursday or so, you're going to find that there's really no dispute about what happened on what date.

Facts are really stubborn things. Facts

are facts. You can't move them, and you can't move the dates on which they occur.

That chronology, the order in which events occurred, is going to demonstrate three really important things to you.

First, you're going to learn that there were three important milestones in the development of antibodies.

Now, Ms. Elderkin suggested that it was Centocor that came up with this mouse antibody -- you remember at the beginning of her discussion -- that was called A2?

Well, mouse antibodies did come first, but Abbott and its scientists had the first mouse antibody in 1986. Centocor did not make the antibody that Ms. Elderkin described until three years later. And I'll come back to that.

You will also learn that chimeric antibodies, part mouse and part human, came second, and that was invented by Centocor but not until several years later.

But more importantly, the first fully human antibody, no mouse parts, no rodent parts, was not made until 1995, 1995, five years after Centocor made its chimeric antibody, one year after this 1994

application that Centocor says told everybody how to make a human antibody.

Now, the evidence will demonstrate to you that this wasn't a simple progression. This wasn't just a natural series of events.

The discovery of a fully human anti-TNF antibody required years of research, completely new technologies, the development of new technologies, and literally billions of dollar to discover and develop and bring to market Humira. It required real innovation. It required real invention.

Now, the second thing the chronology will demonstrate to you is that -- who was first on some of the issues Ms. Elderkin described.

First, it will demonstrate to you, as I said, that the first mouse antibody was actually made by Abbott's predecessors and scientists who ultimately worked for Abbott. Centocor was second.

But most importantly, when we focus on what's involved in this case, a fully human antibody, Abbott was first.

After several years of trying, four years of trying, with some of the greatest scientists in the world, Abbott made what was called D2E7, the active ingredient of Humira in 1995.

In 1996, Abbott filed for a patent application on its Humira.

In 2000, before Centocor even applied for the patent I'm going to show you in a second that's involved here, Abbott had been granted a patent on Humira by the Patent Office.

In 2002, Abbott brought the product to market, and it's been used to treat patients ever since.

Now, third, the evidence is going to demonstrate to you that what Abbott did to develop the fully human antibody was really hard work that required real innovation.

The concepts of hard work and innovation are really important to this case. If you remember in a portion of His Honor's instructions today, he said, when you file a patent application, the patent application has to tell the world how to do it, has to tell the world you can do it, disclose it, but how to make it was His Honor's instruction to you.

Well, the question of whether you told the world how to make it is directly related to how hard was it to make a fully human antibody, how much innovation was required.

If it required an invention by Abbott, if Abbott was first, if it required billions of dollars, it

required years of research, how could it be that Centocor had already taught the world how to do it?

Here, you'll learn from Dr. Salfeld, who's one of the scientists in the courtroom -- if you could stand, Dr. Salfeld -- who's our corporate representative but the scientist who led the team, that our team began in 1991.

It began working with a scientist, a very famous scientist called Dr. Casali. And you'll hear from Dr. Casali. They worked for two years, and they failed.

Then they worked with a different group called Cambridge Auto -- Antibody Technologies for two more years.

As a result of this collaboration, the work that was done by these folks over two additional years, the patent issued. All of the hard work, all of the innovation, all of the creativity is what led to Abbott's patent.

Now, Centocor did decide to make a fully human antibody. Ms. Elderkin mentioned it at the end of her opening. But what she didn't mention to you is, they didn't decide to do this until 1997, after Abbott had been successful, after Abbott had filed its patent application.

Centocor started its fully human project in 1997. And do you know when the product got to the market? Last month, 2009. And I'll come back to that in a second.

It came. They began development after Abbott had been successful. They filed the patent application that's involved in this case after Abbott had been successful. They came to market last month. Now, Ms. Elderkin showed you a slide that compared FDA approval dates. I've added a column. Because what Ms. Elderkin did is, she compared for you approval dates for Remicade, which is the chimeric antibody, and aren't fully human antibodies.

Well, the right comparison is between the two fully human antibodies. And what you'll see is, we were approved in 2002, 2005, 2006. All of their approvals came two months ago.

Now, you will not see or hear any evidence indicating that anyone at Centocor made a human antibody to TNF-alpha or taught anyone else how to do it before Abbott had been successful in 1995.

In fact, what you'll hear is this: You'll hear that Centocor actually tried once, tried once in 1989. It got itself an antibody. Someone else had made it. They brought it in to test it. In fact,

it came from NYU, the other Plaintiff in this case. And guess what? It didn't work. It didn't work.

So by 1994, when they filed this patent application Ms. Elderkin talked to you about, their only experience with fully human was failure.

Now, you may ask, and it's fair for you to ask, if everything that Mr. Lee and Mr. Beck says is true, why are we here?

We're here because Centocor got a patent on Independence Day in 2006. Now, it was filed on July 18, 2002. That patent in your notebooks says right on the face, that's the date that this application was, in fact, filed.

But that date is a problem for Centocor if you consider the instruction His Honor gave you this morning. That date's a problem, because you now know, or you will know from the evidence, by that time, by 2002, Abbott had invented Humira, had made Humira, had patented Humira, and had brought it to market. It was all prior art. It was all prior art.

Now, to be sure, as you will see from the evidence, Centocor was watching Humira very, very carefully.

And you can't see this particularly well now, but you'll see DX233, which will show you that

during this very period of time when Centocor did not have a fully human antibody, it was watching everything that Abbott and its predecessors were doing.

Well, if that's true, if the application was filed in 2002 and Humira was already out there, again, why are we here?

We're here because of what Ms. Elderkin said to you. We're here because Centocor says, well, actually, this application relates back to something we filed in February 1994. It relates back to the application. That application had fully human anti-TNF antibodies.

And so we actually were first. We had it in 1994. But the evidence will show that in 1994, all that Centocor had was chimeric. The evidence will show that all they described was chimeric.

Now, look, there is no dispute that they came up with this chimeric antibody. When you look at the patent, you'll see 13 patent applications on the cover. You'll see that they got patents that cover chimeric antibodies.

Their product is a great product. No one's trying to take that product away from them. No one's trying to take their patents away from them. No one's trying to take the $10 billion in profit that

they've made from it.

To the extent that someone is making a chimeric antibody, they are using their patent, and they should pay. But the question is not about chimeric antibodies. This case is about human antibodies. And I put on the screen Claims 1 and 2, which you're going to hear more than you'll ever want to hear about in the next four days. But as His Honor said, this is what defines what the invention is. And for our purposes right now, I just want to make two points to you.

This claim, the claims that you're going to be asked to focus on, 2, 3, 14, and 15, don't cover a chimeric antibody. They don't cover what Centocor had done. They cover humanized and human antibodies.

Now, as the Court has already instructed you, for that 1994 application to be good, for the patent to be valid, that application has to have described a fully human anti-TNF-alpha antibody, and it must have taught the world how to do it.

It must teach those of ordinary skill in the art, scientists in the field, how to do it. Just sticking the word human antibody in isn't enough. Any lawyer can do that. The specification has to describe more.

Now, this concept of teaching those of ordinary skill in the art will undoubtedly be new to you. It certainly was to me when I looked at my first patent case.

THE COURT: Five minutes.

MR. LEE: What this concept is, is the concept of -- it's probably determined by the old proverb, give a man a fish, you feed him for a day; teach a man how to fish, you feed him for a lifetime. And all laws require that you teach them how to fish. The evidence that you're going to hear on these critical issues is that Centocor's patents have 28 examples. None of them describe human.

Centocor tried to make human and failed. Centocor did not describe how one of ordinary skill in the art could make a fully human antibody.

And you won't have to wonder whether it was hard, because you'll hear the evidence about Abbott's work that led to a fully human antibody. And you're going to hear evidence that when Centocor decided to make a fully human antibody, it took them years and over $300 million.

If what Ms. Elderkin said is true, if Centocor had the invention of a fully human antibody in 1994, why did it take 15 years to take it to market?

Why did it take $300 million? Does that make sense? We will suggest no.

Now, I want to briefly just stress two issues in my remaining two or three minutes.

First, on infringement, Ms. Elderkin referred to tests. What you're going to find on the issue of test is this: On issues on which Centocor had the burden of proof, they did tests. On issues on which we had the burden of proof, we did tests. And each party commented upon the other's tests.

And what you're going to see from those test results is, Centocor did tests in 2007, not under the supervision of its expert. And those tests were done the wrong way, and those tests cannot sustain Centocor's burden of proof.

Finally, on the issue of damages, Centocor suggests to you that it's entitled to $2.2 billion in damages, because Remicade and Humira compete with each other.

Let me just make these four points quickly.

First, you're going to find out that the products are really different. Remicade is administered intravenously. Humira is administered subcutaneously. It's a big difference.

Second, Remicade for rheumatoid arthritis has to be taken with this really, really powerful other drug called Methotrexate. Humira doesn't.

Third, you're going to find from Centocor's own documents that patients perceive Humira as much safer.

And fourth, you're going to see that notwithstanding what Centocor told you today, that fully human antibodies and chimeric antibodies compete, you're going to see that just two months ago, Centocor said to the investing public the opposite.

Now, at the end of the day, we're going to ask for you to find these four patent claims that cover human antibodies invalid. We're going to ask you to find that there's no infringement.

If there's no valid claim, if there's no infringement, then there are no damages.

At the end of the day, we're going to ask you to conclude that Centocor is trying to compete in the courtroom rather than the marketplace, and we're going to ask you to send the parties back to the marketplace where we can compete on innovation, price, and quality.

Thank you.

THE COURT: Thank you, Mr. Lee.

All right. Ladies and Gentlemen, we're going to take a break here in just a few minutes. I want to give you just a few more instructions.

First of all, the lawyers have correctly used some of their exhibits that have already been admitted into evidence. If you see an exhibit that you think you want to -- might want to see at the time when you're deliberating, you need to make a note of that number exhibit in your pad.

Because there's boxes of exhibits, and when I give you my final instructions, I will tell you that I'm not going to send all of those boxes back to you. But I will send back anything you request.

So I'm -- just make a note in your notes if you see something that you think you might want to see later.

Now, ordinarily, we will always break at noon, at 12:00 o'clock, but the Court has another matter that I must take up at 1:00.

So we're going to -- when you come back, we're going to come back in here at 1:20 -- I mean at 10:20, and we will stay till 12:15 today and break from 1:15 till -- or 12:15 to 1:30.

Otherwise, ordinarily, we always break right at 12:00 o'clock.

Remember that this week, I remind you that we will be in trial four days this week, through Thursday, not later than 5:30, and then we'll come back and finish this case early next week.

So I just wanted to remind you of those items.

You may -- let me give you your first break. Be ready to come back in the courtroom at 10:20. Remember the instructions. Certainly, you haven't heard any evidence, but don't start discussing this case among yourselves during these breaks.

You may leave the courtroom at this time.

COURT SECURITY OFFICER: All rise.

(Jury out.)

THE COURT: Please be seated.

The Court is formally in recess, but I want to see counsel up here. But the Court is in recess. I need to see counsel at the bench on the record.

(Bench conference.)

THE COURT: All right. Has anybody got a -- do y'all have -- either side have a shadow jury?

MS. ELDERKIN: We do not.

MR. BECK: We do not.

THE COURT: Oh, okay. Well, I don't ever

want to make comments about -- you know, is there anything that you want to bring to my attention at this stage?

MR. SAYLES: I didn't hear the discussion at the bench when you were talking about -- excuse me -- when you were talking about the arbitration, but there's a stipulation that I intend to read.

THE COURT: Well, the stipulation can be read. What I instructed them on the bench was just to stay with whatever the arbitrator's award said and, of course, any stipulations you got. I can't remember everything that --

MR. SAYLES: Yes, sir.

THE COURT: If you knock him in the head, that's okay, anytime you wish. You have my permission.

MS. ELDERKIN: Does that go for me, too?

THE COURT: No. That's just him. We don't let counsel -- co-counsel can get after it.

MR. BECK: That's called getting my attention, right?

THE COURT: Just let you know that I hadn't forgotten you.

All right. I'll see y'all.

(Recess.)

COURT SECURITY OFFICER: All rise.

(Jury in.)

THE COURT: Please be seated.

Who will be the Plaintiffs' first witness?

MR. SAYLES: May it please the Court.

At this time, we would call Mr. Joe Scodari as the first witness.

THE COURT: All right.

COURTROOM DEPUTY: Raise your right hand, please.

(Witness sworn.)

MR. SAYLES: May it please the Court.

JOSEPH SCODARI, PLAINTIFFS' WITNESS, SWORN

DIRECT EXAMINATION

BY MR. SAYLES:

Q. Would you state your name, please.

A. Yes. My name is Joe Scodari.

Q. Mr. Scodari, would you tell the jury a little bit about yourself?

A. Yes. I'm married; have been married about 35 years. And we have four adult children, two boys and two girls.

Q. And what is your current occupation?

A. Well, currently I'm retired. I retired from Johnson & Johnson a little bit over a year ago in March

of 2008. And I'm currently serving on some Boards of Directors.

Q. Before your retirement, how long did you spend in a career in the pharmaceutical industry?

A. Well, I started at an entry level, as a sales representative, with the industry about 35 years ago. And over the course of the next 35 years, worked my way up to eventually run the pharmaceutical business for Johnson & Johnson.

Q. Your entry-level job, was it what's commonly called a drug rep?

A. Yes, that's correct. My role was to essentially help physicians and nurses and other healthcare professionals about our products, answer any questions that they might have.

Q. And from there, you worked your way through positions to what position when you retired from Johnson & Johnson?

A. Well, I worked my way up over that 35-year period through various management roles, and, ultimately, I was named the Worldwide Chairman of the Johnson & Johnson pharmaceutical business.

As Ms. Elderkin mentioned earlier, Johnson & Johnson is the largest and most diversified healthcare manufacturer in the world, and it's organized in three

sectors: Consumer and personal care. Products that you would know there would be the baby products, shampoo, powder, Tylenol, for example.

Medical devices and diagnostics, and pharmaceuticals.

I was responsible for the pharmaceutical segment in my last position at Johnson & Johnson.

Q. And what was your relationship to Centocor?

A. Well, along the way, as my career developed, in fact, early in 1996, I joined Centocor as its President of the pharmaceutical business. I later was named President and Chief Operating Officer for Centocor.

Q. You mentioned that Johnson & Johnson acquired Centocor. When did that happen?

A. Johnson & Johnson first approached senior management at Centocor about the possibility of acquiring the company in early 1999, and the transaction eventually did close in the fall of that year, 1999.

Q. And most folks have heard of Johnson & Johnson; it's been mentioned.

Just tell us a little bit about Johnson & Johnson.

A. Sure. Well, Johnson & Johnson, as I mentioned, is the largest, most-diversified healthcare

manufacturer in the world. It has a presence in most markets around the world, most countries around the world.

And as I said, has a very diversified business in the sense that there are many consumer products that you would be familiar with, but also is a leading innovator in many areas of -- areas of medicine. And, you know, with respect to the pharmaceutical sector specifically, that segment of the business is involved in the development and commercialization of drugs that really cut across a wide gamut of diseases, from Alzheimer's disease, schizophrenia, infectious disease, autoimmune diseases, cancer.

So it's a very, very diversified business. And, in fact, in pharmaceuticals, Johnson & Johnson is about the fifth largest of all pharmaceutical companies in the industry.

Q. When did you join Centocor?

A. I joined Centocor in April of 1996.

Q. So you were with them before they were acquired by Johnson & Johnson in 1999?

A. Yes, I was.

Q. And then continued on with Johnson & Johnson. And did you have affiliation with Centocor throughout that time?

A. Yes. I decided, as did many members of the management team at Centocor, to stay with the company, even after it had been acquired by J&J. Ultimately, I stayed with the company for about eight years.

There was a period in 2001 to 2003 where my responsibilities did not encompass Centocor. But other than that period, I remained involved with Centocor up to my retirement from Johnson & Johnson last year.

Q. If you could briefly just tell the ladies and gentlemen of the jury what the business of Centocor is.

A. Well, by way of background, Centocor really was amongst the first biotechnology companies ever founded anywhere in the world. The company was founded in 1979, and the real focus of Centocor was to do pioneering work in the area of engineering antibodies to address diseases that the human body would not necessarily on its own create antibodies against.

So from the very beginning, it was that platform, that antibody platform, that served as the focal point for all the R&D, all the commercial activity, all the manufacturing activity that the company did throughout its life and, of course, continues today as a wholly-owned subsidiary of Johnson & Johnson.

Q. When you joined Centocor in 1996, what was

going on in its business at that time?

A. Well, it was a pretty small company at the time. We had a very small diagnostics business which used this antibody technology as well.

And at the time I joined, it had gained its first approval about a year earlier for the first therapeutic use of an antibody. So I joined, as I say, about a year after that product was approved.

Q. And was there a drug in development called Cyntoxin?

A. Well, Cyntoxin actually had failed in development a few years before I joined the company. Cyntoxin was an antibody that was developed to target a very, very serious blood-borne infection called sepsis or septic shock, a very serious disease typically occurs in hospitalized patients.

And at that point and even continuing to today, there are few, if any, treatments that really effectively manage that disease.

So Centocor's first effort to develop a therapeutic antibody was Cyntoxin, and, unfortunately, as often occurs in R&D and in our industry, in the pharmaceutical industry, that product failed in the early '90s.

Q. Are you familiar with that history?

A. Yes, I am.

Q. Let me ask you a specific question.

Are you familiar with the amount of money that was spent in that effort that was not successful?

A. Yes. I would estimate, based on what I know of the history, that the company would have invested somewhere between 4 and $600 million in the development of Cyntoxin before it eventually failed.

Q. And after Cyntoxin failed, what did Centocor do next historically?

A. Well, that led to a pretty tough time for the company, because that was the lead asset that was being developed, and it led to the company substantially restructuring, cutting down the size of the organization. It led to the necessity to make some very difficult resource deployment decisions.

In other words, we had to be very selective, or the company -- before I got there, the company had to be very selective about where it invested its money. The idea being that we wanted to eventually succeed in bringing therapeutic antibodies to the market.

Q. Since you are the first witness, I want to develop a few things that the jury has heard about. First, let's start with what is Remicade.

A. Well, Remicade is the antibody that you heard

previously that was designated as cA2 in its very early development. It is a chimeric antibody, as that has already been described.

And it was designed at a point in time when there was a belief -- there was a belief that this naturally occurring protein, TNF, when it raised or occurred in raised levels in human beings, might be a culprit in a wide range of autoimmune diseases.

It wasn't absolutely clear at the time that Centocor began its work in this area that, in fact, that was a valid target to address those diseases. But there was some good scientific support for believing it might be useful there.

And as a result of that belief, Centocor made the decision to invest in the development of that molecule for those diseases.

Q. And to this day, is Remi -- Remicade an important drug for Centocor?

A. Yes. I must say I am personally particularly proud of Remicade. This drug literally has changed the lives -- and this class of drugs, I have to say, has changed the lives of patients affected by a wide range of autoimmune diseases.

We have heard about two of them already this morning: Crohn's disease and rheumatoid arthritis.

As of today, Remicade has really helped benefit literally hundreds of thousands of patients. And, in turn, it's also created a very attractive and successful business for Centocor and for Johnson & Johnson.

Q. Has it received approvals for the treatment of specific diseases?

A. Yes, it has. In fact, its first approval was in the fall of 1998 for Crohn's disease, and then about a year later was approved for rheumatoid arthritis.

Q. And we say approval. Are we talking about the Food & Drug Administration, the FDA?

A. Yes. Any drug that is developed for ultimate commercial sale in the United States goes through a very rigorous process. And there are very specific defined guidance that is provided by an agency of the federal government called the Food & Drug Administration. I will refer to that by FDA -- or shorthand, FDA.

And the FDA typically works to regulate sponsors who wish to bring new therapies into the market. That work really starts with the discovery of the molecule, will involve laboratory testing in the early days, can involve animal testing. And eventually, with the consent of the Food & Drug Administration, a sponsor can begin what's called clinical testing or testing of the molecule in human beings.

And only after those tests have determined that the molecule is effective for the indication that's being studied and safe for that indication does the FDA grant an approval for that sponsor to bring that molecule into the marketplace.

Q. I want to stop for a moment and ask you if you saw Plaintiffs' Exhibit 254 that was shown during the opening, which was the Centocor marketing document relating to the rating of overall safety.

Did you see that?

A. I did.

Q. First of all, based on your knowledge, experience, and background, is Remicade a safe drug?

A. It certainly is.

Q. You saw Plaintiffs' Exhibit 254. From where did that statement come that was shown to the jury in opening?

A. Well, what the exhibit was speaking to was perceptions of customers or potential customers, physicians in the marketplace, about the various anti-TNF agents that compete in that market.

So that information is strictly that; it's perceptions. The reality is that the FDA --

Q. Let me stop you right there.

Was that a Centocor market research report?

A. Yes, it was.

Q. All right. And with respect to safety, does the FDA have any requirements that they impose before a company can make a claim about safety of one drug over another or any safety?

A. Yes, they do. In fact, it would be inappropriate at this point in time to suggest that there's any comparative difference between Humira and Remicade with respect to either efficacy or safety, because the FDA provides very specific guidance to the industry with respect to what needs to be done before a statement like that can be made.

And very specifically, what they require is that there need to be what are called two; so two separate studies, adequate, and well-controlled. So well-designed studies that meet certain statistical requirements that demonstrate, in fact, that there are some differences, whether they be the effectiveness of the drug or the safety of the drug.

No such studies have ever been conducted involving both Humira and Remicade, and, therefore, neither company can make a statement of comparative efficacy or safety at this point in time.

Q. You indicated earlier that Remicade was approved for the treatment of rheumatoid arthritis, and

I would like you to tell the jury what that disease is briefly, please.

A. Well, I think actually the Abbott legal representative showed an x-ray, which I think does a reasonable job of depicting what can happen in this disease. As has been mentioned earlier today, there are two different types of arthritis.

Osteoarthritis, you can think about -- that's the one we commonly think about. You can think about that as being more of a mechanical disease. It's sort of the wearing of the joints that eventually leads to pain and inflammation. And that disease can be treated with antiinflammatories to reduce the pain.

Rheumatoid arthritis is an autoimmune disease. So this is a situation where a naturally occurring substance in the body that should be there -- TNF in this case -- begins to appear in human beings at much higher levels than are normal.

And when that occurs, it begins to attack the host, attack the human being who has that elevated level. So in rheumatoid arthritis, what happens is TNF levels get very escalated. It begins to attack the joints, and over a period of time, patients with rheumatoid arthritis can eventually become substantially physically disabled.

There's tremendous -- and you can see that in that x-ray. You can see tremendous damage to the joints. So a very, very debilitating disease; a disease that dramatically negatively affects patients and their ability to live a normal and high-quality life.

Q. Were there other early clinical results with cA2, which became the antibody known as Remicade?

A. Yes. The early work that was done, again, in this very resource-constrained environment after Cyntoxin failed, focused in two disease areas. One was Crohn's disease, and I would say there were two subsets under that umbrella. One was the severity of the disease, moderate to severe Crohn's disease, and also a subset called fistulizing Crohn's disease.

Now, fistulizing Crohn's disease is a really, really terrible disorder. It's -- as I say, it's a subset of Crohn's, which is an inflammatory bowel disease. But when a patient develops fistulizing Crohn's disease, open lesions from the bowel to the outside world can occur, and you can imagine how that can impact a patient's quality of life.

Before Remicade was studied in that indication, the only solution to that disease was surgical. To our tremendous surprise, when we studied the drug in that application, it was the first drug ever

demonstrated to close fistula non-surgically.

So those were two indications that we developed in addition to rheumatoid arthritis.

Q. So in short, Crohn's disease is a very serious bowel disorder?

A. That's correct.

Q. And after getting these initial results in the testing of cA2 that became Remicade in rheumatoid arthritis and Crohn's disease, what did Centocor do?

A. Well, we were challenged, I would say, from the standpoint of available resources at this time, because, again, this was while Centocor was still an independent company. We had very limited resources, but we did make the decision at that time to invest some of those limited resources in the investigation of the drug's potential utility in Crohn's disease and in rheumatoid arthritis.

Q. What investment was needed?

A. Well, similar to what happened with Cyntoxin, actually.

By the time Remicade was initially approved by the FDA here in the United States, in the fall of 1998, the company would have invested by that point somewhere between 4 and $600 million in that drug, in R&D, in manufacturing capability, and in getting ready to bring

that product to the marketplace.

Q. Is the process for getting FDA approval lengthy?

A. It can be. As I say, the FDA outline some very specific guidance that pharmaceutical company or biotechnology company sponsors must follow. Those guidelines are very rigorously determined to ensure that before a drug becomes available in this country, to the extent that it's possible, that we understand as much as possible about its potential benefits and its potential risks.

Q. Can it take years?

A. It can take many years.

Q. And is there ever any assurance along the way of the FDA approval process that you will indeed get approval at the end?

A. No. And, in fact, the best example of that, quite frankly, is Cyntoxin. That drug was in very, very late-stage clinical development. Hundreds of millions of dollars had already been invested in it, and, ultimately, it failed in late-stage clinical development. And that product goes away after that failure.

So this underscores how risky it is to be an innovator and a pioneer when developing a new technology

for a very difficult-to-treat disease.

Q. You told us about these early clinical results with cA2. That was before FDA approval; is that right?

A. That's correct.

Q. And once you got those early clinical results that you've mentioned in Crohn's and RA, wasn't this a safe investment?

A. No, not really. Again, we -- we were excited, because what we were seeing in these early studies, early human studies, was remarkable efficacy. But it was understood that these were very early studies, typically in very small numbers of patients.

And we understood that it was a very long road to meet the FDA's requirements to bring such an interesting possibility, let's say, to the marketplace. So absolutely no assurance at any point along the way that we would, in fact, you know, gain FDA approval.

Q. Here we are in June of 2009. Did it turn out to be a good investment?

A. Well, it did. And obviously, a lot of things look really good in retrospect. And I have to say it's one of the areas that I take great pride in in my career, to look back and say we made some very difficult decisions when we didn't have a lot of data or facts. But we did invest appropriately in Remicade, and it has

been a very, very successful treatment for hundreds of thousands of patients. It's literally changed their lives. And I would often get phone calls from patients thanking us for what we did to develop Remicade. And it's also turned out -- now, I always used to say that our business was about doing well as a business as a result of doing good for patients. And I can't, frankly, think of a better example than Remicade of that -- of that idea.

We have dramatically benefited patients, but we've also built a very successful business as a result of that.

Q. You were on board with Centocor in 1998 when it received its first approval for Crohn's.

A. That's correct.

Q. And you were on board with Centocor in 1999 when it received its first FDA approval for rheumatoid arthritis?

A. That's correct.

Q. Was there publicity surrounding those approvals of that drug?

A. Yes, there was. There was very substantial publicity. And I should also mention that when the drug was approved initially for Crohn's disease, in 1998, one of the major patient advocacy groups here in this

country called the Organization for Rare Disorders -- National Organization for Rare Disorders awarded Remicade its prize in that year as the most innovative new medicine for a rare disease.

So there was tremendous publicity both at the time it was approved for Crohn's disease, which acknowledged the fact that this was a breakthrough drug, and also a year later, when it was approved for rheumatoid arthritis.

Q. And to the extent that you haven't already, what was the reaction of the pharmaceutical industry to these approvals?

A. Well, as I mentioned, it's often uncertain whether or not a given target, in this case TNF, can be successfully treated with a given drug, such as Remicade.

So when a company in the industry, in this case Centocor, is able to demonstrate in human trials that the drug has dramatic benefits for patients and also has an acceptable safety profile, the industry typically takes notice of that, and, you know, recognizes that, you know, that there may be an opportunity to bring other competitive agents into that area, once it's understood that that target, in fact, can be positively impacted by the first drug in that

category.

Q. You saw during the opening that counsel held up the Galien Prize that they received in 2007.

A. Yes.

Q. Did Remicade receive the Galien Prize?

A. Yes, we did.

Q. When?

A. It was several years before that. I don't remember the exact year. I think it was around 2004, but it was a number of years before Humira received the award.

And, truthfully, both products deserve that award. They were both dramatic innovations that have really benefited patients around the world.

Q. Does the Galien Prize have anything to do with patents and patent rights?

A. No, it doesn't.

Q. Approximately how much did Centocor spend to develop Remicade and conduct clinical trials through FDA approval?

A. Up until that initial approval, right? So this is now 1998. We would have invested somewhere in the range of 4 to $600 million to get Remicade to that -- to that position.

We've invested substantially more than that

since 1998.

Q. You were with Centocor when it was bought out by Johnson & Johnson?

A. Yes, I was.

Q. Would you tell the ladies and gentlemen of the jury how it came about that Johnson & Johnson purchased Centocor?

A. Yes. A Vice Chairman of Johnson & Johnson, one of the most senior people in the firm, approached us in early 1999, approached our CEO, Chief Executive Officer, in early 1999, indicating that they were very enthusiastic about the early results that had been seen with Remicade, and that they believed that they could provide substantially more investment capital to advance that product and subsequent products to the market.

So, in essence, they made a proposal that they would acquire the company with the idea that they could invest more than what we might be able to invest as an independent company.

Q. Were you directly involved, on behalf of Centocor, in the process that led up to Johnson & Johnson purchasing Centocor?

A. Yes, I was.

Q. And what was the purchase price that Johnson & Johnson paid to acquire Centocor?

A. It was just under $5 billion, which at the time, was the largest single acquisition Johnson & Johnson had ever made.

Q. Were there any other companies expressing an interest in Centocor at that time?

A. What happened was around the spring of 1999, so as the discussions were still underway with Johnson & Johnson, some rumors surfaced that Centocor might be the subject of a takeover.

When that appeared in the public press, other companies did, in fact, express interest in acquiring the company.

Q. Was Abbott one of those?

A. Yes, it was.

Q. Did you personally have discussions with Abbott personnel about acquiring Centocor?

A. Yes, I did.

Q. And with whom did you meet and discuss the possible acquisition of Centocor by Abbott?

A. There were two people involved in those discussions on both sides. In the case of Centocor, it was Mr. Holveck, Dave Holveck, the CEO, and myself. And for Abbott, it was Miles White, who was the CEO of Abbott at the time and remains their CEO, and Arthur Higgins, who ran their pharmaceutical business at that

time. He has since left Abbott.

Q. And were you personally involved in discussion with those Abbott gentlemen?

A. Yes, I was.

Q. Did Abbott's Chairman tell you why they were even interested in acquiring Centocor in the first place?

A. Well, their interest essentially was the same as Johnson & Johnson's interest. They recognized, based on the early data and the initial approval of Remicade and Crohn's disease, that this could be a very, very interesting opportunity, could tremendously benefit many, many patients, and, in turn, generate a very interesting business opportunity.

Q. Did Abbott get to the point of actually making a formal offer for Centocor?

A. To my recollection, there was never a formal offer made. However, they did float numbers that were substantially below what Johnson & Johnson was willing to pay and what we believed represented fair value for the company.

Q. All right. And once the acquisition was made by Johnson & Johnson, you stayed on board?

A. I did.

Q. And did you continue to, in your job, watch

the competition, see what they were doing?

A. Absolutely.

Q. Do you keep up with Abbott?

A. I do, absolutely.

Q. Did you keep up with Abbott at the time?

A. Absolutely.

Q. And did Abbott make an acquisition based on your knowledge after Johnson & Johnson acquired Centocor?

A. Yes. About 18 to 24 months after we had been acquired, Centocor had been acquired by J&J, the chemical company called BASF, which had a pharmaceutical business under its broad umbrella, called Noel, made the decision to exit the pharmaceutical business and determined to seek a buyer for that business.

Abbott was the company that eventually acquired that business.

Q. And they were working on the TNF antibody?

A. That's correct.

Q. Now, you know that in this case Centocor is charging that Abbott's Humira product infringes the patent-in-suit.

A. That's correct.

Q. And just tell the ladies and gentlemen of the jury briefly your understanding of what is Humira.

A. Well, Humira is also a monoclonal antibody. Its intent or its objective is identical to Remicade's objective. In other words, to bind to TNF in the patient's bloodstream, remove that TNF from the body, and as a result of reducing TNF levels in the body, have a beneficial effect in treating autoimmune diseases, such as Crohn's and RA and others.

Q. And giving credit where credit is due, has Humira been a successful product in the marketplace?

A. Yes, it has.

Q. Can these drugs that we're talking about in this case, these antibody drugs, be expensive to the patient?

A. They can be. You know, the biggest contrast between biotechnology-derived drugs, drugs that are typically derived at their outset from living cells, and most of the drugs that we think about commonly, pills or tablets, is that pills or tablets are based on chemicals.

Whereas these drugs, these biotechnology drugs, are the outgrowth of bioengineering, living cells, to eventually create a drug from them. That process of getting to that point can be very expensive. And, very importantly -- and this is a big distinction of biotech products and chemically based

drugs. The manufacturing investments to bring these products to the market can be very, very, very significant.

Q. Does Centocor have any programs to assist patients who can't afford these drugs?

A. Yes, we do. We recognize that we have a responsibility to patients who may have a difficult time paying for these very expensive medications. And, in fact, we offer -- Centocor offers two programs.

One is a program that makes the drug available for free to patients that have household earnings level below a certain number. And we also offer a program to patients that do have drug benefits but have benefits such that they have to, out of their pocket, pay a very large co-pay. So we have a co-pay assistance program that helps defray that expense for those patients.

Q. And based on your knowledge in the industry, does Abbott have similar programs?

A. Yes, they do.

Q. Now, I want to shift gears with you for a moment.

Did there come a time when Centocor focused on commercially developing a human anti-TNF antibody to treat rheumatoid arthritis and Crohn's disease?

A. Yes, we did.

Q. And what is that drug?

A. That drug, in fact, was just recently approved a couple of months ago. The generic name is golimumab and the brand name is Simponi.

Q. Can we just stick with Simponi?

A. We certainly can.

Q. All right. And whose decision was it to develop Simponi?

A. Well, I actually led that decision when I was the President and Chief Operating Officer at Centocor.

Q. And when did work on Simponi begin?

A. We made the decision to begin the early work on that molecule in 19 -- late 1997.

Q. Now, from 1997 -- and you said it came out a few months ago in 2009.

What took so long?

A. Well, I mean, I think the Simponi example really underscores how much time, how much effort, how much investment, and how much risk a company must take to successfully bring a molecule such as this to the marketplace.

Q. And whose decision was it to focus those resources on Remicade in commercial development before focusing on Simponi?

A. Yeah. Again, let's remember this is all

happening in the period before Johnson & Johnson had acquired the company.

We were very resource-constrained. I should mention the company didn't make its first profit -- although it was founded in 1979, we didn't make our first profit as a company until the fourth quarter of 1997.

So as you might imagine in that environment, we had to be very careful about where we invested resources. And although we recognized that a human antibody could be interesting, the cA2 antibody was further advanced, and we made the decision at that time, because of resource constraints, to focus our investment there.

Q. All right. I want to change topics with you again at this point.

Based on your business experience of 35 years in the pharmaceutical industry, how important are patents to pharmaceutical companies?

A. The truth is that without patents, it would be very difficult for the -- for the industry to be successful. These are products, again, that take many, many years and literally hundreds of millions of dollars to develop.

And if one goes back to the comments His Honor

made at the beginning of the trial, patents are issued in order to protect the innovator for a period of time, not forever, but for a period of time, so that they are incented to make those big investments, take those big risks.

Q. And was it Centocor's practice, while you were there, to ever engage in licensing discussions with actual competitors?

A. Yes.

Q. Why would you do that?

A. Well, you know, when -- when one is making these decisions to invest these hundreds of millions of dollars, one wants to be certain that when that product eventually does arrive on the market, there is, in fact, the ability to have the freedom to commercialize those products.

And so in those circumstances -- and they do occur from time to time -- where competitors may have intellectual property or patents that we believe are either critically necessary or possibly necessary, we will typically seek out a license to that patent. And we will pay fair value when we license those patents.

Q. And sometimes you'll license the company's technology, Centocor's or Johnson & Johnson's, to another competitor.

A. Yes, that's correct.

Q. Why do you do that?

A. Well, again, you know, the circumstances in the development of biotechnology products often lead to multiple patents being required in order to advance the development of a drug.

And sometimes the reason that those kinds of decisions are made is that both companies in those discussions need something from the other company. So it's not infrequent that those kinds of negotiations can lead to what's called cross-licensing, where each company provides a license to each other's technology.

Q. Is it fair to say that as a company you're willing to do that because what goes around, comes around?

A. To some extent.

Q. Now, in this case, Plaintiffs' Exhibit 1 is the '775 patent, which is in the jury's book. And they've seen it, and I'm going to put it up on the screen here.

Do you recognize this as the '775 patent?

We're not going to go through all the terms, I promise you.

A. Thank you.

Yes, I do.

Q. Were you made aware of the official notification by the Patent & Trademark Office that the claims of the '775 were going to be allowed?

A. Yes, I was.

Q. And about when did that official notice come?

A. That was in December of 2005.

Q. Did Centocor ever offer to license the '775 patent to Abbott?

A. Yes, we did.

Q. When?

A. In December of 2005.

Q. In what context?

A. Well, we anticipated -- of course, you can't absolutely predict these things, but we did anticipate that the Patent Office would eventually advise us that they would allow the patent and ultimately grant the patent.

So in the period before that occurred, we did a couple of things. Number one, we took a look at whether or not products in the marketplace, including Humira, might infringe that patent. And we did certain experiments to determine whether or not Humira infringed the patent.

Those experiments documented that, in fact, it does.

And so as a result of that, we also discussed extensively how we might tell Abbott about that. And a decision was made to communicate to them essentially, immediately after we received this notice of allowance, that we were going to be granted this patent and that we believed it read on Humira; in other words, that Humira would infringe it, that we had tested that and confirmed it infringed.

And as a result of that, we believed that Abbott should consider taking a license to it.

Q. Tell us who participated in those discussions.

A. The people that were involved in those discussions were two from the Abbott side and two from the Johnson & Johnson side.

On the J&J, or Johnson & Johnson side, there was myself and my head of Business Development, a gentleman by the name of Tom Heyman.

And for Abbott, it was two individuals on their side; one is a Mr. Bill Dempsey. He was, if you like, my direct counterpart at Abbott. He was responsible for the pharmaceutical business at Abbott. And as well, his business development person, a gentleman by the name of John Poulos.

Q. Did you personally have discussions with Mr. Dempsey at Abbott about this patent?

A. Yes, I did.

Q. How long did those discussions -- how long a period of time did those discussions span?

A. From the time that we first advised Abbott that we had received this notice of allowance until the time that we ultimately made the decision to file suit, that period ran from December 2005 until April of 2007. And we had a number of meetings. From time to time, there would be periods where we'd have a number of successive meetings. Then there would be periods where there were very few meetings. But over that period from time to time, we had meetings on the topic.

Q. Did you ask Abbott to pay for its use of the '775 patent?

A. Yes, we did.

Q. At any time did they ever agree to pay for their use of the '775 patent?

A. No, they did not.

Q. In your discussions with Mr. Dempsey, did you say to him that it was Centocor's belief that Abbott infringed the '775 patent on the claims that had been officially allowed?

A. Yes, I did.

Q. I want you to look at Plaintiffs' Exhibit 161 in evidence. I've actually put a copy on the podium for

you there to save some time. And the first thing I want to do, when I get this up here, is make it bigger. And let's -- it's from you, Joe Scodari, and it's dated March the 12th of 2006, right?

A. That's correct.

Q. And this is to a list of people. I'm not going to ask you to name them, but just tell the jury who you were sending this communication to.

A. So, generally speaking, when I was involved in these kinds of discussions with a third party, I would typically write notes that would inform other members of my staff or of management of the progress of those discussions. And this is what this e-mail was intended to do.

Q. Let's highlight the first sentence that says: I received a call late Friday from Bill Dempsey.

So is this memorializing an actual telephone call between you and Mr. Dempsey of Abbott?

A. Yes, it is.

Q. Now, let's go down to the third paragraph, and I want to focus on this sentence that says: He characterized the discussion as one in which our view was not fully recognizing the value enforceability of their patents, and, once again, raised the non-enablement argument on the TNF patent.

Let me stop right there. In the context of your discussion with Bill Dempsey, what was the TNF patent that's referred to in your memo?

A. This is the TNF patent that we just discussed that was granted to Centocor.

Q. It says: Once again, he raised the non-enablement argument.

Had you discussed this with Mr. Dempsey before?

A. From the very beginning of our discussions back in December of 2005, once their patent people had taken a look at this patent, their feedback to us was that they believed -- or they did not believe -- let's put it that way -- that the patent was enabled.

Q. Did you consistently tell him that you believed that they infringed?

A. Yes, we did.

Q. And did you say that to Mr. Dempsey?

A. Yes.

Q. I want to go to the sentence that says: I told him that we felt strongly about the quality of the TNF patent, and given the fact that Humira is in the market is an issue they needed to take seriously.

In context of your discussion with Mr. Dempsey, would you tell us what this means?

A. Well, what it means is that we believed that our invention, the reference to the human antibody and the methodology for producing that antibody, was a very, very valuable piece of intellectual property.

And as a result of that value, we had indicated to Abbott on numerous occasions that we needed to be appropriately, fairly compensated for their use of that technology.

Q. Then the next sentence that begins he indicated: He indicated that they were taking the patent seriously and recognized that they can be challenged on infringement but felt their position was defensible.

Do you see that?

A. Yes, I do.

Q. In context, tell us what was being discussed between you on the one hand and Mr. Dempsey on the other.

A. Well, during the course of these conversations, which, again, occurred on and off between December 2005 and April 2007, we were never able to really get a specific proposal from Abbott as to how they would compensate us for their use of this patent or this intellectual property.

So in this particular telephone conversation,

which was one of a number of either telephone conversations or meetings, I made the point to him that since we had not really gotten any feedback that suggested they were oriented toward compensating us for that patent, that they needed to take it seriously. And he acknowledged that, in fact, they were taking it seriously.

He also acknowledged that they could be challenged with respect to infringement of the patent but reinforced that their issue with the patent was non-enablement.

Q. Now, we have an e-mail here that's dated March of 2006.

Did you have discussions of this nature with Mr. Dempsey before this?

A. Yes.

Q. Did you have discussions with Mr. Dempsey after this?

A. Yes.

Q. And in any of those conversations you had with Mr. Dempsey up until the time that this suit was filed, did Abbott ever offer to pay anything for the use of these patents?

A. No, they did not.

Q. Did Abbott continue to sell Humira anyway?

A. Yes.

Q. And has Centocor or Johnson & Johnson ever received a nickel for the sales of Humira that infringe this patent?

A. No, we have not.

MR. SAYLES: I'll pass the witness.

Your Honor, I'm going to offer the version of 161 into evidence that we displayed. We have agreed with counsel that it is admissible.

It's got unredacted portions following our pretrial conference, but it is agreed. And that was the proper exhibit that I showed him. It's all in. And I offer it.

MR. BECK: That is correct, Your Honor. We have no objection.

THE COURT: All right. That's 171?

MR. SAYLES: 161.

THE COURT: 161 is received. Thank you.

MR. LEE: Your Honor, we have a notebook of the actual exhibit for the witness in case he'd like to see it in addition to what's on the screen.

May we provide it to the witness?

THE COURT: Certainly.

MR. LEE: And we have a copy for the clerk and for Your Honor, if you want it.

MR. SAYLES: Mr. Lee, do you happen to have one for me?

MR. LEE: Yeah, we have one for you as well. I'm sorry, Mr. Sayles.

THE COURT: Give that to the clerk. Thank you.

MR. LEE: May I proceed, Your Honor?

THE COURT: You may.

CROSS-EXAMINATION

BY MR. LEE:

Q. Good morning, Mr. Scodari.

A. Good morning.

Q. Mr. Scodari, I want to help the jury with the chronology of events a little bit.

You were here this morning when Ms. Elderkin did her opening, correct?

A. Yes, that's correct.

Q. And you were here when she said February 1994 is a very important date in this case, correct?

A. Yes, she said that.

Q. Now, you didn't even join Centocor until 1996, correct?

A. That's correct.

Q. So to the extent that February 1994 was an important date, and we need to know what was going at

Centocor then, we're going to ask someone else, correct?

A. I would say it would be appropriate to do that. I would also say that in order to effectively conduct my duties as Chief Operating Officer, I had to understand the history of the company.

Q. But you weren't there.

A. I was not there.

Q. Now, you're not trained as a scientist, correct?

A. That's correct.

Q. Have you yourself ever made a mouse anti-TNF-alpha antibody?

A. I have not.

Q. Have you yourself ever made a chimeric anti-TNF-alpha antibody?

A. I have not.

Q. Have you yourself ever made a fully human TNF-alpha antibody?

A. I have not, but I should add that over the many years of managing businesses, high-technology businesses, one needs to really take seriously the on-the-job training that comes along with those roles and those responsibilities.

Q. Mr. Scodari, my question was, did you ever make one.

A. I said no.
Q. And you surely have never made a fully human high-affinity neutralizing anti-TNF-alpha antibody, correct?
A. I have never personally done that.
Q. Now, you mentioned the '775 patent a few minutes ago, correct?
A. Yes.
Q. Have you read it?
A. I have not read it in detail.
Q. Well, you haven't even read the patent itself?
A. I said I hadn't read it in detail. I have read it, not in detail.
Q. Have you read the claims that the jury is going to be asked to make a decision on?
A. Yes.
Q. All right. So you've read Claims 2, 3, 14, and 15, correct?
A. That's correct.
Q. Now, you've testified at length today about Remicade, correct?
A. That's correct.
Q. Remicade is a chimeric antibody, correct?
A. That's correct.
Q. By the time you arrived at Centocor, Remicade

had been developed, correct?

A. No, that's not correct. It was in development, but it had not been developed.

Q. Fair enough.

The antibody, A2, had been developed before you arrived at Centocor, correct?

A. A2 had been identified as a promising antibody candidate, yes.

Q. A2 was a mouse antibody, correct?

A. That's correct.

Q. And it was made in 1989, was it not?

A. I don't know the exact date, but if you're saying that's when it was, I'll support that.

Q. Does that sound about right to you? I mean, you said you familiarized yourself with what had been going on before.

A. Absolutely, but I don't remember the specific date.

Q. But you know it happened before you arrived in 1986 (sic), correct?

A. I arrived in 1996. Yes, correct.

Q. And you know that before Centocor identified the mouse antibody, whenever it did it, someone named Dr. Moller had already made a mouse antibody and published on it, correct?

A. I do not know that.
Q. You have no idea whether that's true or not?
A. I don't.
Q. Okay. But you do know that cA2, a chimeric antibody, was in development when you arrived in 1996, correct?
A. That is correct.
Q. And you do know that the chimeric antibody had human parts, correct?
A. Yes.
Q. And you do know that the chimeric antibody had mouse parts, correct?
A. That's correct.
Q. And, in fact, Remicade is about 25 percent mouse, is it not?
A. I don't know the exact percentage.
Q. All right. Well, can you tell the jury this: I want you to focus on Claims 2, 3, 14, and 15 in their notebooks.

You said you've read those claims, correct?
A. Yes.
Q. Those claims don't even cover Remicade, do they?
A. I can't tell you that without looking at the document.

Would you like me to look at that?

Q. Sure.

Turn in your notebook to PX1, and we'll put it on the screen.

MR. SAYLES: Excuse me, Your Honor. I am going to object to that on two grounds. One is, he's asking a question that I did not go into on direct; and, second, he is attempting to compare the Remicade product of Centocor in defense of the claim of infringement as opposed to comparing it to the claims.

So I object to it on that basis.

THE COURT: Well, I'll overrule your first objection.

What do you say about that, Mr. Lee? You're trying to compare different products. I mean, that's not what the jury is going to be asked to do.

MR. LEE: I agree with that fully, Your Honor. I think that the jury just heard about 40 minutes about Remicade and the chimeric antibody, and the question of whether that's covered by the claim or not --

THE COURT: I disagree with you. I don't see the relevancy here, so I'm going to sustain that objection.

MR. LEE: All right.

Q. (By Mr. Lee) You have read Claims 2, 3, 14, and 15, correct?

A. Yes, that's correct.

Q. And you understood them, correct?

A. To the extent that a non-patent attorney can understand them, yes.

Q. Sure.

A. I knew enough about them to ask the right questions.

Q. Now let's get us all on the same page. If cA2 was in development in 1996, when did you have cA2? What's the date?

A. I don't know what you mean by that question.

Q. Well, when is the first time Centocor had made chimeric antibody?

A. Well, the initial chimeric antibody occurred after the work that was done in conjunction with Centocor by NYU.

Q. Okay.

A. I don't know exactly what that date was.

Q. Well, can you help us fill out the chronology? When is it that NYU or Centocor first had cA2?

A. I can't. I don't know that date.

Q. Okay. And you can't tell us whether it was before 1994 or after?

A. I cannot.

Q. All right. Now, you did know that -- you did tell the jury that Centocor started a project involving a fully human antibody, correct?

A. That's correct.

Q. Now, that did start after you arrived, correct?

A. That's correct.

Q. That was 1997?

A. I believe it was late 1997, correct.

Q. Let's put up here late 1997, Centocor starts fully human anti-TNF-alpha antibody, right?

A. Correct.

Q. Now, before late 1997, Centocor had not had a project to make a fully human anti-TNF-alpha antibody, correct?

A. Not to my knowledge.

Q. But before 1997 -- in fact, before 1994, Centocor had had this product called Cyntoxin, correct?

A. Correct.

Q. Cyntoxin was a fully human antibody, correct?

A. I don't think that is correct actually.

Q. You don't know -- do you know one way or another?

A. I'm not positive, no.

Q. In any event, Cyntoxin was an antibody, correct?

A. That's correct.

Q. And it had failed, correct?

A. That's correct.

Q. At any time -- based upon the information you acquired through the company, at any time before February of 1994, had Centocor made a fully human anti-TNF-alpha antibody?

A. I don't know what happened before 1994, but my understanding is no.

Q. Okay. At any time before 1997, had Centocor even made a fully human anti-TNF-alpha antibody?

A. No.

Q. So the first time it started was in 1997. And when were you successful?

A. I don't remember exactly when the decision was made as to which antibody would move into clinical development, but it would have been a number of years after 1997.

Q. And how many more years?

A. I don't know.

Q. But you do know it came to market; it got FDA approval in 19 -- in 2009, correct?

A. That is correct.

Q. All right. So let's just be sure that we have this portion correct.

If I ask you to focus on Centocor's chimera antibody projects, that was underway when you arrived, correct?

A. If you're referring to cA2, yes.

Q. All right. And it continued after you arrived, correct?

A. That's correct.

Q. And you were successful in bringing a product to market when?

A. In the fall of 1998.

Q. So the fall 1998 is when cA2, or Remicade, comes to market, right?

A. That's correct.

Q. Now, if we focus on the fully human antibody, as far as you know, nothing was done before 1997, correct?

A. Not by Centocor.

Q. Right. It had been done by others, correct?

A. I'm not sure of that.

Q. Well, you told Mr. Sayles that you followed on what was going in the industry, correct?

A. Yes.

Q. You knew that BASF had a project involving

anti-TNF-alpha antibodies, didn't you?

A. Yeah, I did. Yes, that's correct.

Q. And you know that before 1997, BASF had been successful, correct?

A. I don't know exactly when BASF, or Noel, had actually achieved or made the decision to advance the antibody that later became known as Humira.

I do know that Noel was, in fact, working in this space during my tenure at Centocor.

Q. You know that Noel was working on anti-TNF-alpha antibodies before you even started your project, correct?

A. Yes, that's correct.

Q. And the general community, people interested in these products, knew before you started your project that Noel was working in this area, correct?

A. Yes, that's correct.

Q. And you knew that they had successfully made a fully human anti-TNF-alpha antibody before you started your project, correct?

A. Again, I don't remember the exact timing of when the decision was made to advance the molecule that later became Humira.

Q. But I'm not asking you about the exact time, and I apologize if I was unclear.

Was it before or after you started your project?

A. It was before we started our project.

Q. Right. So before you had ever started your project, you knew that BASF had been successful in making a fully human antibody, correct?

A. That's correct.

Q. And, in fact, that occurred after 1994 and before 1997, correct?

A. Again, I can't be specific on the dates, because I just don't know the specific dates.

Q. All right. Fair enough.

Now, when the project started in 1997, you were at Centocor, correct?

A. That's correct.

Q. And you had a group of scientists working on the project, correct?

A. That is correct.

Q. And you invested a substantial amount of money in developing Simponi, correct?

A. Over a period of time, yes. In the early days, somewhat limited, but later, substantially more.

Q. Well, if we take the numbers you were talking about with Mr. Sayles, over a period of time, you invested about $300 million in developing and bringing

to market Simponi, correct?

A. I don't know where you got the 300-million number.

Q. Does that sound low or high?

A. I don't know. It's probably in the ballpark from the start of the project until the initial FDA approval, but I don't actually know exactly what that number is.

Q. Well, let me ask you to apply the same standard you applied when you gave Mr. Sayles estimates of what it costs to bring some of your drugs to market.

Judging by the same standards, about how much did it cost to bring Simponi to market after you started it in 1997?

A. I couldn't guesstimate the exact number. The only thing I would say is because it was then known that this was a viable target, much of the early work that would have been done with Remicade would not have been necessary with a successor molecule, such as Humira or -- or the human antibody project that was started at Centocor.

Q. So the answer is, you can't give me an approximate amount.

A. I would say it's probably in the range of 4 to $600 million, but I don't know where in that range the

number is.

Q. All right. Now, we can agree that however much was spent was spent between 1997 and 2009 to bring a fully human antibody to market, correct?

A. That's correct.

Q. And you were here in the opening when Ms. Elderkin said that you actually had the invention of a fully human antibody back in 1994, correct?

A. That's correct.

Q. So it took you 15 years after the date on which you had the invention of a fully human antibody to bring it to market; is that correct?

A. That's correct.

Q. Okay. Now, you talked about some licensing discussions with Abbott, correct?

A. Yes, I did.

Q. Now, the first set of discussions you talked about were discussions that you had with Abbott at about the time that Johnson & Johnson purchased you, correct?

A. That's correct.

Q. And those occurred around 1999, correct?

A. In the spring of 1999, yes.

Q. Right. And what happened is, Abbott talked to you, a company that had a chimeric antibody, correct?

A. That's correct.

Q. And it talked to BASF, a company that had a fully human antibody, correct?

A. Not at the same time. They spoke to us in the spring of '99. The discussions they would have had with Noel would have happened sometime in 2001.

Q. Right. And after having talked to both companies over a period of a couple of years, they decided to acquire a company that had developed the fully human anti-TNF-alpha antibody, correct?

A. I think that's a characterization of what happened.

What actually happened was that they were extremely interested in acquiring Centocor. They were not willing to be competitive with respect to how much to pay for the company, and later, when they didn't successful acquire Centocor, remaining very interested in this target area, blocking TNF, when the Noel opportunity became available, they then moved to acquire that company.

It wasn't a decision they made to pick one or the other, because the timeframes were very, very different.

Q. In any event, what they did is, they acquired the company that had developed already a fully human anti-TNF-alpha antibody and brought it to market,

correct?

A. That's actually not correct. If my memory serves me correctly, Humira was approved after they acquired Noel.

So it wasn't fully --

Q. That was what I said. I apologize if it was unclear.

I said they had decided to acquire the company that had developed a fully human anti-TNF-alpha antibody and then bring that product to market; is that correct?

A. Yell, I guess where I'm getting hung up is the word develop. And I should say that the process of drug discovery and develop starts with discovery, early development.

Developed means you've successfully finished all the human trials and brought it to them. So developed in the past tense means it's done.

Q. Okay.

A. It wasn't done when they acquired the company.

Q. Well, let's use a different -- let's use a different set of words.

They decided to acquire a company that had made a fully human anti-TNF-alpha antibody, correct?

A. That is correct.

Q. They decided to invest in that fully human

anti-TNF-alpha antibody, correct?

A. That is correct.

Q. They decided to bring it to market, correct?

A. That's correct.

Q. And they did, correct?

A. That is correct.

Q. And to quote you, you described Humira as a dramatic innovation, correct?

A. That's correct.

Q. And it was, was it not?

A. What I said was that all of the drugs that block TNF were and are dramatic innovations, because before those drugs became available, these patients had incredible difficulty trying to manage disease, and they've made a tremendous difference.

Q. Remicade is a dramatic innovation, correct?

A. That's correct.

Q. Humira is a dramatic innovation, correct?

A. Humira is an innovation that built on the innovation that had already been established by Centocor with the chimeric antibody.

Q. Mr. Scodari, weren't your words just 20 minutes ago, it was a dramatic innovation?

A. I don't -- you'll have to read the record to me. I don't remember exactly what I said, but I don't

dispute the fact that these are both very innovative molecules.

However, I think it's also important to understand that in the realm of drug development, when one sponsor effectively blazes the path --

MR. LEE: Your Honor, could we ask the witness to answer --

THE COURT: Sustained. What you need to do is answer the question that he asks.

THE WITNESS: Okay.

THE COURT: You got to realize, Mr. Sayles, if he wants to clear something up for the jury, he gets to come back and ask questions to clear up anything.

So try and limit your answers to the question asked, okay?

THE WITNESS: My apologies.

Q. (By Mr. Lee) Now, Mr. Scodari, let's go to these discussions you had with Abbott at about the time you found out you were going to get a patent.

Do you have those in mind?

A. Now, which discussions? We're talking about the TNF patent discussions.

Q. The TNF patent discussions.

A. Yes. Yes.

Q. And you said they occurred between a period of 2005 and 2007, correct?

A. December 2005 to April of 2007, correct.

Q. Now, let me ask you about some specifics. You told the jury that you had test results that would indicate that Abbott was infringing, correct?

A. That's correct.

Q. Abbott asked you for those test results, correct?

A. That's correct.

Q. You refused to give Abbott those test results, correct?

A. As far as I know, that is correct.

Q. Right. So that when you accused Abbott of infringing and Abbott said, show us the proof, you said, not going to show it to you, right?

A. That was the decision that our patent department made, yes.

Q. Right. And that's a decision that you abided by, correct?

A. That's correct.

Q. Now, Johnson & Johnson is a big company, as you told us, correct?

A. That is correct.

Q. It gets many letters from patent-holders that

say, We have a patent; we think you're infringing; you should pay us, correct?

A. I don't know. I'm not in the Patent Department, so...

Q. Do you know whether Johnson & Johnson gets letters from patent owners that say Johnson & Johnson needs to take a license from it?

A. I don't know that personally.

Q. It's never happened to you during the time that you were the head of the Pharmaceutical Division?

A. I've never gotten a letter from an inventor saying, I have this invention; we want you pay us for it, no.

Q. All right. Well, then Mr. Dow is going to testify. He's one of the patent lawyers, correct?

A. That's correct.

Q. When Johnson & Johnson gets a letter from another company saying that there is a patent and you should take a license, what you do is, you ask your folks to look at the patent, correct?

A. Yes.

Q. You make a decision as to whether the patent is valid, correct?

A. Yes.

Q. You make a decision as to whether the patent's

infringed, correct?

A. Yes.

Q. And if you decide that Johnson & Johnson is not doing anything wrong, the patent's invalid, the patent's not infringed, you decide -- you tell them we're not going to pay you, right?

A. That's correct.

Q. And that's what a responsible company should do. It should decide -- it should look at the patent, it should decide what it covers, and then it should make a decision as to whether it should pay, correct?

A. That's correct.

Q. Simply because someone comes and says, I have a patent; you should pay, wouldn't lead you to pay, correct?

A. No, not without digging into the patent.

Q. Right. Let's bring up PX161, could we, which is the exhibit that Mr. Sayles asked you about earlier. Do you have that?

A. Yes. Yes, I do.

MR. BECK: Can y'all see that with this?

MR. LEE: We can move this.

MR. BECK: You want to move it?

MR. LEE: Now, could we blow up the third sentence of the third paragraph?

Q. (By Mr. Lee) Now, this paragraph, this third sentence refers to the non-enablement discussion that the parties had, correct?

A. Yes, that's correct.

Q. And you discussed this with Mr. Sayles, correct?

A. Discussed it with Mr. Sayles?

Q. The discussions between the two of you about the issue of enablement, correct?

A. You mean Mr. --

Q. Mr. Sayles asked you -- I apologize.

Mr. Sayles asked you some questions about discussions you had with Abbott, correct?

A. That's correct.

Q. And during those discussions, you discussed the question of enablement, correct?

A. The issue of enablement was raised by Abbott as --

Q. Right.

A. -- or the lack of enablement was raised by Abbott as the reason they didn't believe that they needed a license to the patent.

Q. Right. So from the beginning, Abbott said to you, This patent is not enabled, correct?

A. That is correct.

Q. And you understood what -- in general terms, what enablement is, correct?

A. Yes, that's correct.

Q. Enablement is the requirement that the patent teach people of ordinary skill in the art how to make the invention, correct?

A. That's correct.

Q. And what Abbott told you is, since you're accusing fully human antibodies of infringing, you didn't teach people how to make and use fully human antibodies.

That's what they were telling you, correct?

A. That was Abbott's position, correct.

Q. And that has been consistently Abbott's position since 2005, correct?

A. That's correct.

Q. Now, you disagree, correct?

A. That is correct.

Q. Right. And the purpose of this proceeding is to determine who's correct, right?

A. Not on the question necessarily of enablement, but primarily on the question of whether there's infringement.

Q. Well, you understood that Abbott contended the patent was not enabled in 2005, correct?

A. That is correct.

Q. And you were here for my opening in 2009, correct?

A. That's correct.

Q. And I said it's not enabled today, correct?

A. That's what you said.

Q. Now, at any time prior to filing this lawsuit, did you ever send anybody a letter at Abbott -- did you ever send anybody at Abbott a letter that said, You're infringing our patent?

A. I don't know whether Johnson & Johnson, as a company, ever did that. I did not personally do that.

Q. Did you ever send to Abbott a letter that says, Your Humira product infringes the claims of these patents?

A. No. But we personally discussed that with both Mr. Dempsey and Mr. Poulos.

MR. LEE: Well, Your Honor --

THE COURT: Sustained. Please limit your answers to the question asked.

Q. (By Mr. Lee) At any point, after doing these test results you discussed and before filing a lawsuit, did you say, Abbott, we'll share with you the reasons why we think that you infringe this patent?

A. Not to my knowledge. I certainly did not do

that. I don't know whether Ken Dow did that.

Q. At any -- but that's a decision that your lawyers made, correct?

A. That's correct.

Q. All right.

MR. LEE: Nothing further, Your Honor.

THE COURT: Mr. Sayles?

REDIRECT EXAMINATION

BY MR. SAYLES:

Q. As a businessman with Centocor and Johnson & Johnson, did you have access to the Legal Department?

A. Yes, I did.

Q. And in the context of these discussions, did Abbott's lawyer call your lawyer?

A. Yes.

Q. All right. You were asked a number of questions by Mr. Lee about the development of the fully human antibody.

Do you recall that line of questions?

A. Yes, I do.

Q. When you were answering him, would you tell the ladies and gentlemen what you meant by development?

A. Well, development really begins from the selection of the antibody. So that's sort of in the early discovery phase right through the eventual FDA

approval of the drug for its intended use.

Q. And you were asked a number of questions about whether you had a project on fully human antibodies.

Do you remember that line of questions?

A. Yes, I do.

Q. And when you talk about a project as a businessman, are you talking about commercializing a product?

A. Eventually, yes. That would be the goal.

Q. And when you were talking to Mr. Lee about a project, is that what you were talking about, the commercial development of an item, in this case, Simponi?

A. Yes.

Q. As a businessman, are you aware that companies can obtain patents on inventions for which they never have a commercial product?

A. Oh, absolutely. It happens all the time.

Q. You were asked some questions about FDA approval. As a businessman, is it your understanding that FDA approval has anything to do with the governmental agency, the PTO, in the issuance of patents on inventions?

A. No, not at all.

Q. You were asked some questions about -- from --

on Simponi, that it took 15 years to bring Simponi to the market.

A. Yes, that's correct.

Q. Do you recall that?

A. Yes.

Q. In answering Mr. Lee, did your answers have anything to do with whether Simponi had been invented at an earlier date?

A. No.

Q. What were you talking about?

A. I was talking about the development of the molecule.

Q. A commercial development?

A. That's correct.

Q. That does seem like a long time. In the pharmaceutical industry, is 15 years a long time to commercialize a product?

A. It's not unusual in our industry to have that length of time from the start of a project to the eventual commercial availability of that product in the marketplace.

Q. And you were asked some questions about whether you ever caused a letter to be sent to representatives of Abbott charging them with infringement.

Do you remember that line of questions?

A. I do.

Q. Did you personally say to Mr. Dempsey, the head man at Abbott, that you thought they were infringing the '775 patent?

A. I did.

Q. Did you personally say to Mr. Dempsey that you expected them to pay for that use?

A. Yes, I did.

MR. SAYLES: I'll pass the witness.

MR. LEE: Your Honor, just one question.

THE COURT: All right.

RECROSS-EXAMINATION

BY MR. LEE:

Q. Mr. Scodari, whatever your definition is of development, what is the date on which you first made Simponi; you first had it as a compound?

A. I don't know. The only thing I can tell you is it was after 1997 when we started the project.

MR. LEE: Nothing further, Your Honor.

MR. SAYLES: I have nothing further of this witness, Your Honor.

THE COURT: You may step down.

THE WITNESS: Thank you.

MR. SAYLES: May he be excused, Your

Honor?

THE COURT: Any objection to excusing the witness?

MR. LEE: None, Your Honor.

THE COURT: Okay. You're -- you may be excused.

Who will be your next witness?

MR. SAYLES: May it please the Court.

At this time, we would call Mr. William Dempsey by deposition. And this is the only one that has to be read, and it's very short.

THE COURT: All right.

MR. SAYLES: And, Your Honor, may I read the questions and the answers, since it is short, rather than having a reader?

THE COURT: However you would like to do it.

MR. SAYLES: All right. We've agreed on an introduction of William Dempsey, and it's as follows:

Mr. Dempsey is a former business executive for Abbott. Mr. Dempsey worked for Abbott from April of 1982 through August of 2007.

In 2005, Mr. Dempsey was Senior Vice President for Abbott's pharmaceutical operations and was responsible for the U.S. pharmaceutical business. He

was responsible for the commercial aspects of Humira.

Beginning in 2006 and until retired from Abbott in 2007, Mr. Dempsey was the Executive Vice President of the Global Pharmaceutical Products Group.

And I will read the questions and the answers. After Mr. Dempsey was duly sworn, with counsel for both parties present, he testified as follows:

QUESTION: I'll take you to the February 2006 timeframe.

Do you recall anyone from J&J or Centocor telling you that Centocor had a recently allowed patent that covered TNF-alpha antibodies?

ANSWER: Yes.

QUESTION: What do you recall about that conversation?

ANSWER: That particular meeting, John Poulos and I had a meeting with Tom Heyman and Joe Scodari to advance in negotiations we had going on with a variety of issues, some of which, maybe all of which I previously referenced.

The purpose of the meeting was to work out a proposal to try and find a mutually acceptable -- acceptable resolution of the issues.

We got to the meeting, and this was at the J&J, New Brunswick, if I remember correctly, and we

went up to the conference room, and Joe started out the meeting by saying something along the lines of, gee, I hate to blindside you, or I'm sorry I didn't give you a heads-up or something like that, but we just had a TNF patent allowed. You may want to consider or evaluate the context of what we're discussing here.

That's my recollection.

QUESTION: Did Mr. Scodari say anything else about the TNF patent?

ANSWER: Not that I can recall specifically, no.

QUESTION: Do you recall him saying that he thought that Abbott's Humira product infringed the claims of the TNF patent?

ANSWER: I don't recall that.

QUESTION: Do you recall if he said anything -- if you said anything to Mr. Scodari about the issue?

ANSWER: My recollection is, I said we'll have to evaluate this and take a look at it.

QUESTION: Do you recall anyone from J&J or Centocor ever telling you that they viewed the TNF patents as strong patents or as valuable technology?

ANSWER: The way it was characterized to me is, this is something that they thought was important

that we needed to consider.

QUESTION: Did you disagree with that?

ANSWER: I have no basis for making any sort of judgment. I wasn't familiar with it.

QUESTION: Did you ever express to anyone at Centocor and J&J that you disagreed with their assessment?

ANSWER: Yes.

QUESTION: What did you tell them?

ANSWER: I thought the patents weren't very strong, and we weren't concerned about them.

QUESTION: And who specifically did you tell that to?

ANSWER: Joe Scodari.

MR. SAYLES: That concludes Mr. Dempsey.

THE COURT: Who will be your next witness?

MR. SAYLES: At this time, our next item of proof, Your Honor, would be to read to the jury Abbott's response to Request to Admission No. 13.

And may I tell them or ask the Court to tell them what a request for admission is?

THE COURT: Well, a request for admission is one party, such as, in this case, Centocor sent to the Defendant, Abbott, and requested them to admit or

deny certain facts.

And if they admit certain facts, those are deemed conclusively proven.

So you may go ahead.

MR. SAYLES: This is Request for Admission No. 13.

Admit that on or about December 13th, 2005, a representative of Johnson & Johnson and/or Centocor informed Abbott that a notice of allowance was received from the United States Patent & Trademark Office for the claims that issued as the '775 patent.

Response: Admitted.

That concludes this portion of the request for admissions, Your Honor.

THE COURT: Okay. Have you got a witness?

MS. ELDERKIN: May it please the Court. The Plaintiffs call John Ghrayeb.

COURTROOM DEPUTY: Raise your right hand, please.

(Witness sworn)

MS. ELDERKIN: Set to go?

THE WITNESS: Yes.

JOHN GHRAYEB, Ph.D., PLAINTIFFS' WITNESS, SWORN

DIRECT EXAMINATION

BY MS. ELDERKIN:

Q. Would you please introduce yourself to the jury.

A. My name is John Ghrayeb. I'll give you a little --

Q. Tell them a little bit about yourself.

A. -- background about myself. You may be wondering about my accent. I was born in Israel to a Christian Arab family.

When I was 16 years old, I was offered a scholarship to finish my high school in England, which I did. Then I went to Oxford University, got my first Bachelor of Science degree in chemistry.

Then came to the United States, went to Kent State University in Ohio and received my Ph.D. in biochemistry. That's where I met my wife, also.

Then I spent two years supported by a National Institute of Health grant on a first doctoral training where I learned many techniques in molecular biology. And then I joined Centocor in 1984.

Q. And are you still at Centocor?

A. No, I'm not.

Q. When did you retire?

A. I retired in September of 2006, and I've, you know, been sort of active in the field since then.

Q. Okay. What is your relationship to the patent in this lawsuit?

A. I am an inventor on this patent.

Q. Okay. So what kind of work did you do for Centocor while you were employed there?

A. When I was hired by Centocor, they were looking for somebody with expertise in recombinant DNA protein expression, and I spent my entire career working on antibody engineering and antibody production and different aspects of that technology.

Q. How many different products did you work on while you were at Centocor?

A. I worked at many projects while I was at Centocor, but I was -- I'm happy to say by that -- to date, four of these products have been approved. So I'm sort of very happy to have made that achievement.

Q. And when you say approved, you mean approved by the Food & Drug Administration?

A. That's correct.

Q. So they --

A. Or -- or -- or the European authorities.

Q. So there are four different products that you worked on that either are on the market now for patients or shortly will be?

A. Yes. Yes.

Q. Just briefly, since you said you worked in the field of antibodies during your career, can you explain to the jury, what is an antibody?

A. Okay. An antibody is a natural substance, it's a protein, that your body produces to defend itself against foreign invaders, if you like, in your body. Every day while even you're sitting, you know, your skin is exposed to halogens, to all kinds of things, bacteria, and the body has this great system called the immune system that is always looking for these things that are not supposed to be there.

So among their defenses are these proteins known as antibodies. So if you were to get a vaccine, you know, you -- they give you a version of that virus or the protein that you might be exposed to, and your body then makes antibodies against it.

And the beauty of it is, it remembers. So the next time you get that same infection, it quickly gets rid of it. And as was said earlier, you might not even realize that you got the flu or whatever it was because you had the immunity to it.

Q. Did you work with us to prepare a slide to help explain the structure of antibodies?

A. Yes, I have.

Q. Okay.

MS. ELDERKIN: Can we put that up, please?

Q. (By Ms. Elderkin) And I think you have a laser pointer there with you.

Could you explain for the jury what's depicted on this slide, please?

A. Okay. These are two representations of what an antibody may look like. I have to say that, you know, nobody's seen it inside the human, so these are based on structural analysis.

The one on the right is a very complex version of the antibody protein, but what it tells you is that it's made up of those building blocks called amino acids that you heard about earlier.

So to make it easier -- and by the way, that's how all scientists represent antibodies when they do publications.

We'll concentrate on this picture. So you see they've been color coded for clarity.

So each antibody has what's known as a light chain. You see here it's light, because it's small. And it's also made up of a heavy chain, which is called heavy because it's bigger.

What up here -- I think it's in purple -- represents what are known as the variable regions. Now,

these compromise the part of the antibody that is responsible for binding or latching on to whatever target. If it's a virus, whatever it is that it's trying to find, that's the business end, if you like, of the antibody.

It's not to say that the rest of this, which is known as a heavy chain, is not important. It has many other functions to help the immune system get rid of whatever is there.

So the important thing to remember is that the variable regions are really what determines what the antibody is.

The other parts are often known as constant regions, because many antibodies have the same so-called, as mentioned, heavy chain or constant regions.

But every one of the millions of antibodies that may be in your blood at this point, you know, will have a different set of variable regions. That's how you can recognize so many different proteins.

Q. Dr. Ghrayeb, you mentioned amino acid building blocks. Can you explain what you mean by that?

A. Now, the power proteins made in your body -- I mean, DNA, which is present in all of us, is the way that the body can remember what type of proteins to

make. So in that DNA, there is instructions on how to make the proteins.

And the proteins, when you look at them, it looks very complicated, but it's really very simple, because it's all made of 20 different building blocks known as amino acids.

The way they are put together, the way the DNA instructs the cell to make the protein is simply how these amino acids are put together, what order, and how many of them are there.

So you have small proteins, large proteins. But these are very important. And it doesn't matter what organism, whether it's mouse, human, rabbit, these same building blocks are used.

Q. So how does a mouse antibody differ from a human antibody?

A. The mouse antibody is based on DNA that is found inside the mouse.

The human antibody is based on DNA that may have been obtained from a human.

Q. But they're all made from the same 20 amino acids.

A. Absolutely, yes.

Q. There's nothing different in a mouse antibody than is different -- than is in a human antibody with

respect to the building blocks that they're made of.

A. The chemical composition would be the same.

Q. Now, before we get back into the technology, let's go back to Centocor when you joined it in 1984. What was the company like then?

A. I mean, what attracted me to Centocor was -- compared to a larger company that I interviewed with, was that it was small. When I joined, there were about a hundred people.

It was very focused on this technology, and there was a lot of energy and excitement. The main drive is to get things into humans and -- as quickly as possible.

So it was a very exciting time to, you know, start a career at Centocor.

Q. Now, there was some previous testimony -- I don't think you were in the courtroom -- about a drug that Centocor was developing called Cyntoxin. What was Cyntoxin?

A. Cyntoxin was a human IgM antibody. If -- as I mentioned on this slide, these constant regions come in different varieties. So there is one variety called IgM. This antibody was developed to treat a very complex disease called septic shock. Now, the cause of sepsis shock is usually a bacterial

infection in the blood. These are the worst kind of infections that you can get. And it's often -- in a large percentage, it could lead to death, because you get organ failure and so on.

So this drug was developed to try and fight this disease.

Q. Okay. And by what date did Centocor have Cyntoxin, the human antibody for sepsis?

A. I want to say before 1990. It was in the '80s.

Q. Okay. Why didn't Centocor take Cyntoxin to market?

A. This is the -- what people don't usually appreciate about developing products, is you can invent a new product; you can find it; you spend all this time putting in the clinic; but until you treat human patients with it, you won't know how good it's going to be.

So with Cyntoxin, what the company did is, it took it into clinical trials, and we did do one reasonably large but not very large clinical trial where the results were very promising.

So we went to the FDA and asked for approval. The FDA, upon looking at the data, said, you know, it looks really promising, but we would like you to do

another larger trial.

So we did that. Unfortunately, we could not replicate the results.

And this is a very common feature of development. I mean, you can go so far, and then at the end, you may not be able to get approval, because that's how it is.

Q. Did Cyntoxin harm anybody?

A. No.

Q. Was it a bad antibody?

A. No.

Q. Was it a failure, as a scientific endeavor, to develop a human antibody, to discover a human antibody?

A. Absolutely not. I think the -- it's really the science that -- wasn't applied correctly to the human. In fact, there was a mouse antibody that was being developed by a competitor of ours that also failed in the clinic.

So it really was the target that -- or the understanding of the disease. Not the reagent, not the drug that caused the antibody -- I wouldn't want to call it failure -- not to be approved.

Q. Right. And Cyntoxin was not an antibody for TNF, right?

A. No, not at all.

Q. And to this day, years later, has anybody been able to develop an antibody to treat sepsis?

A. Not to my knowledge.

Q. So what was your work at Centocor towards developing antibody therapeutics or antibody drugs?

A. Right. Our mission -- and, you know, the management was very clear. The company started in the field to develop antibodies. Our mission is to try and make antibodies that would be suitable for use in humans as quickly as we can.

So we -- we use any available, you know, technology that's available to take antibodies, like mouse antibodies or whatever antibodies were available, and make them suitable so we can use them in humans.

Q. Okay. And did you sometimes start your projects with mouse antibodies?

A. Yes, we did.

Q. And what did you do with the mouse antibodies?

A. So the -- with the mouse antibody, we used what are called recombinant DNA techniques, and you got an introduction to it this morning.

Basically, what you do is, you know, you look at the mouse antibody. We already knew at the time that giving that to a human multiple times may not cause harm, but it makes the immune system look at this as a

foreign protein and then try and get rid of it. So the drug won't be any use after a few doses.

So the -- what we tried to do is make the antibody, you know, as human as possible. And in that figure again that's still on the screen, what you can do is substantially change a large part of this protein to make it human.

So, in essence, what you do is you go and find, using the recombinant techniques, you find those parts of the antibody that, as I said, are called the business end.

And then what you do is you cut and paste, you know, just to describe something you're all familiar with. You take these pieces that came from the mouse DNA, and then you splice it to pieces of DNA that came from a human.

So you've now formed something that is much more acceptable when you inject it into a human being.

Q. And do those techniques of being able to cut and splice different pieces of DNA, do they apply whether it's mouse DNA and human DNA or DNA from any other sources, including two sources of human DNA? You know -- right. The technique of splicing DNA is, you know, just taking one DNA from one source and then attaching it to DNA of another source. It doesn't

matter, you know, which -- what species they came from.

Q. And is there a name for an antibody like this where you have DNA that's partly from one source and DNA partly from another source?

A. Yes. That antibody is called a chimeric antibody.

Q. And, Dr. Ghrayeb, in the notebook in front of you, there's a copy of Plaintiff's Exhibit 1. That's also in the juror notebooks. That's the '775 patent-in-suit.

A. Yes.

Q. And you are an inventor on this, correct?

A. Yes.

Q. Okay. And would you tell us, please --

MS. ELDERKIN: And we'll ask Mr. Ficocello to highlight the title of the patent.

Q. (By Ms. Elderkin) What is the title of your patent, please.

A. The title of the patent is recombinant A2-specific TNF-alpha-specific antibodies.

Q. Okay.

MS. ELDERKIN: Then if we can highlight the inventors on the front page, please.

Q. (By Ms. Elderkin) And could you tell the jury, please, who are the other people who are listed as

inventors with you on this patent?

A. They're Junming Le known to his friends as Jimmy Le, Jan Vilcek -- they're both from New York University -- Peter Dadonna, of course, myself, David Knight, and Scott Siegel. They're all -- were at Centocor at the time.

Q. Okay. Perhaps the jury might be curious about the order of the names on that patent.

Is there any rhyme or reason to why they're listed in this order?

A. No. They're listed by institution first, so the NYU was listed first, and then it was alphabetical, and then the Centocor inventors were listed in alphabetical order.

Q. There's somebody else who's not listed here, who I think the jury may hear from -- about later in the trial, Han Trinh. Who is Han Trinh?

A. Han was a technician that worked in the lab that I was responsible for, and her job was to follow instructions given to her by supervisors to work on, you know, different projects, different recombinant DNA cloning projects as directed by her supervisor.

Q. Did she have a Ph.D. degree?

A. No, she did not.

Q. Okay. Now, David Knight is listed as one of

the inventors. What was David Knight's role in this invention?

A. David Knight reported to me, and Dave Knight and I worked on the strategy on the invention of -- of this product by, you know, designing the way we were going to make the final product.

Q. And did he take direction from you with respect to what projects he would work on?

A. Yes, he did.

Q. And during the time period in the early '90s, say up until February of 1994, did you ever direct Mr. Knight to work on a project to make a human antibody --

A. No.

Q. -- in the lab?

A. No.

Q. Why is that?

A. Based on our experience -- two years before we started working on this, we had made other chimeric antibodies. One of them we actually gave to patients. Now, the background is that the mouse version of this antibody was given to patients, and just as predicted -- these were cancer patients -- just as predicted, after multiple doses, the antibody wasn't effective.

So we made a mouse/human chimeric version of

it, and it was given to patients. And the -- there was no immune response measured in the trial.

In other words, the antibody given to the patient even multiple times behaved as you would expect it to. It didn't get removed by the immune system, and it really, to us, said that this was sufficient to make that change.

The body has been -- whatever you want to call it -- adaptive enough to think -- to see that the major part of the protein is human, and it accepted it and -- without clearing it from the circulation or treating it as, you know, some foreign protein.

Q. So your prior work had shown that chimeric antibodies could work very well as drugs for long-term treatments?

A. Correct.

Q. Now, was there a particular project that led to the work that's described in your patent?

A. Right.

Q. What was that work? What was the project?

A. Right. Now, tumor necrosis factor, which is the target for the antibody, you know, has been reported to be important in a variety of diseases, infectious diseases, inflammatory diseases.

And we were very interested in looking for the

right type of antibody, the right type of product that we can use to treat those different diseases. So that was the impetus is, you know, we needed to find a drug to treat those diseases.

Q. Okay. And I take it you worked with New York University on this project?

A. That's right. We had fine experience working with Dr. Le and Dr. Vilcek on other projects where they have -- they made mouse antibodies. And they also -- Dr. Vilcek is very well known -- both of them are known in the whole cytokine field.

So we went and asked them to make an antibody for us that would bind to TNF.

It's important to note that, you know, we were a very small group, and, you know, we often collaborated with the outside in order to get to where we went -- where we wanted to get, you know, as quickly as possible.

Q. So did you follow what Dr. Le and Dr. Vilcek were doing on the project?

A. Yes.

Q. Okay. What was their role in the project, this TNF project?

A. Right. So what Dr. Le and Dr. Vilcek, what they did is they took the human TNF, injected it into

mice. So, obviously, the mice see this human TNF as a foreign protein, and they make antibodies to it.

The project was made, you know, more challenging because TNF is harmful, as you've heard, and even to mice, if you give human TNF, I mean, they can get sick. They can also die if you give too much. So it took, you know, some skill to make the antibody and, you know, not harm the mice at the same time. So once the mice started making antibodies to TNF, using a variety of techniques, they were able to take those cells inside the mouse that are like the factories making antibodies, and then take them -- strip them out of the mouse and allow them to be -- stay alive in a test tube in the lab.

Now, these cells now are producing many, many different antibodies, because in your blood, you are making millions of antibodies. The task then is to find that needle in the haystack and get those cells that are producing the antibody that you want.

So they succeeded. They found different antibodies that bound to TNF and then chose what they perceived to be, you know, the best one.

Q. And what was the best one?

A. The best one they designated as A2. And that -- that -- that one, and I think we looked at

others, were sent down to Centocor for us to evaluate further in many different assays to confirm that, you know, what we really wanted.

Q. And once Dr. Vilcek and Dr. Le at NYU isolated A2, did they have any further role in the project?

A. I would say after that, the development, making the antibody more suitable for human use, was all done exclusively at Centocor.

Q. All right.

A. Now, we kept in touch, but they did not contribute themselves.

Q. Okay. And could you explain again to the jury, how did you make the A2 antibody more suitable for human use in long-term treatment?

A. So what we did is, we took those cells that were sent to us by Dr. Vilcek, and then we isolated, we took the DNA out of these cells. And then for the same reason there is -- the DNA can make millions, thousands of proteins. We have to find the one that is responsible for making the antibody we want.

So we use a variety of techniques, and we isolated that part of the DNA -- that part of the DNA from the A2 cell line that's responsible for the binding, the variable region of the light chain and the heavy chain as I showed you earlier.

Once we identified those, we used more recombinant DNA techniques to attach those variable regions to human constant regions to make -- and then we put those DNA back into another cell.

And the cell now use that new book, if you like, to make an antibody based on that new piece of DNA that we combined and it started making the cA2, which was the antibody that became, eventually, our drug.

Q. So in summarizing that, is it correct to say that you took DNA from a mouse, the instruction booklet part of the mouse and part of the instruction booklet from the human DNA, and you made a new instruction booklet combining those two pieces of DNA, put that DNA in a cell, and that cell then started as a factory to make the new cA2 antibody?

A. I couldn't have said it better.

Q. Ah, I got it from you, Doctor.

Now, there's been some -- there's been some testimony or statements about mouse parts in chimeric antibodies. Would you explain to the jury, is it really a mouse part in the chimeric antibody?

A. I think the -- the key part of the antibody is the part that it binds to. And I think we have to keep remembering that that's the real invention, is finding the antibody that, you know, is -- binds to that TNF in

such a way it never lets it go, and it stops it from working.

Now, that -- that binding side, that variable region, could come from any source. It happens that we -- we made that from a mouse DNA source.

Q. Okay. But if we used the term -- if somebody were to use the term mouse part, it's only because the amino acid building blocks are arranged in a way that the mouse DNA says to arrange it, not because there's anything other than --

A. Oh, no, no. That is not the -- no.

Q. I'm sorry. Don't let me put words in your mouth here.

A. Sorry. No. There's no part of the mouse in the antibody.

Q. Okay. And we -- and there's also been reference to a fully human antibody. Is there such a thing as a recombinant fully human antibody?

A. You know, again, my opinion is -- you know, what's a fully human antibody? A fully human antibody is -- one, if I took your blood right now, and I took some of it out, to me, that's a fully human antibody. If you have to manipulate, you know, the antibody that you make, even though originally it came from a human source, from DNA.

You have to spend some time, years maybe, you know, manipulating it because it wasn't as good as you want. To me, that no longer is a fully human antibody, and that's my opinion.

Q. So the recombinant human antibodies that we're talking about in this lawsuit are made from different pieces of human DNA, correct?

A. Yes.

Q. But the recombinant human DNAs we're talking about in this lawsuit don't appear in nature in anybody's body, right?

A. The original source of the DNA may have come from a volunteer's blood, but the -- in many cases, the researchers have to then manipulate it. They have to mutate it to make it what they want it.

So now they change some of those building blocks in the -- in the sequence. And, you know, to me, how can you say it's fully human? I mean, you have engineered something artificially into those sequences. It will still be a great drug, but I don't see it as fully human.

Q. Okay. How did cA2, the chimeric antibody that you made, compare to A2, the mouse antibody from NYU, in terms of how it bound to TNF?

A. It was identical.

Q. Okay. Let's go back and look at your patent, if we could.

What role did you play in writing this patent? It's a pretty long document.

A. Right. The way, you know, these documents are put together is -- obviously, the inventors or scientists know all the work that went into making the invention.

So it's up to us, all the people on the invention, to put together in writing in great detail their part in that invention.

So what we -- what we did is, we all collaborated to write what eventually became a manuscript and was published as our description in great detail of how we made the cA2.

Then this was given to the lawyers, and then, you know, the lawyers put it together in the right format and figured out what needs to be done and then filed it with the Patent Office.

Q. Did you review and sign off on this, though, before it was filed in the Patent Office?

A. Yes, I did.

Q. Okay. Do you have other patents in addition to this '775 patent-in-suit?

A. I think the last time -- I may have about 60

different patents on various subjects.

Q. So in your patents, generally, is it unusual that your patent covers an invention that's broader than what you actually did in laboratory experiments?

MR. LEE: I object, Your Honor. What these other patents have -- what these other patents --

THE COURT: Overruled. I'm going to allow it.

MR. LEE: Okay.

Q. (By Ms. Elderkin) Would you like the question again, Dr. Ghrayeb?

A. Yes.

Q. In your experience with your other patents, is it common or uncommon that the patent may actually claim or cover something that's broader than what you actually did in the lab, the experiments that you actually did?

A. Yeah, that's correct.

Q. And do you know why that's the case?

A. It -- I think what was -- when you first make the discovery, as the time goes by, you make more discoveries.

And then as a scientist, you don't always see all the value of what you -- I mean, we can -- some of us tend to be humble, and then, you know, with the advice of others and good patent attorneys, we can also

understand that, you know, what we have is much broader than what we even had dreamt of.

But I do believe that the invention that we made, the key part of it is, you know, we discovered the drug that was extremely effective and, you know, can be applied to any sort of -- any drug that would, you know, target the same TNF.

Q. Okay. Now, on the first page of your patent here, there's a section that says related U.S. application data.

MS. ELDERKIN: And you might need to pull up the page to get the whole section in there, if you would, Mr. Ficocello.

If you could highlight that, please, or enlarge it.

Q. (By Ms. Elderkin) Do you have an understanding of what this is, this related U.S. application date, Dr. Ghrayeb?

A. Yes. This goes through all the applications that were filed from the first date to, you know, the current date and all the individual applications that were sent to the Patent Office to amplify, to, you know, increase the information about the invention.

Q. Okay. And are these all related in some way?

A. Yes, they are.

Q. How are they related?

A. Well, they're all continuations on, you know, other patents with more information to make it, you know, current and then to expand, you know, the scope of, you know, the information that was provided.

Q. Okay. And why? Why did you do that? Why did you file so many applications?

A. I think it was important to -- as more information was -- was -- became available on the invention, that you disclose it. And as more techniques became available for other people to use to make the invention, that you make sure that it's disclosed to the Patent Office.

Q. How many patents have issued on this series of patent applications, in addition to the patent-in-suit?

A. I think there are about six.

Q. Six? Okay.

And in your personal experience with your other patents or patents that you've overseen as a research director at Centocor, how unusual is it to have a series of applications that might span over 10 or more years like this, a series of related applications?

A. It's not unusual.

Q. And it's not -- why is it not unusual?

A. Because it -- you know, the whole process of

providing all the necessary information and then keeping it updated and, you know, working with the Examiner of the Patent Office, that -- that's a long process, and it's not unusual for it to take that long.

MS. ELDERKIN: Your Honor, before we get into the meat of the patent, would this be a good time to break?

THE COURT: Sounds good to me. You can talk me into about a minute-early lunch.

Ladies and Gentlemen, if you recall, I told you earlier this morning we were going to break now, and I want you to be ready to come back in the jury room at 1:30, 1:30.

And keep in mind my instruction about not discussing the matter.

You may leave the courtroom.

COURT SECURITY OFFICER: All rise for the jury.

(Jury out.)

THE COURT: You can step down. Everyone step down.

I've got a sentencing matter to take up at 1:00 o'clock, so you might fold everything up. You don't have to remove it from the courtroom, but, you know, just sort of stack it, because we're going to take

up a matter at -- a criminal sentencing at 1:00 o'clock, and hopefully, I'll be through by 1:30.

Got anybody in the audience that needs to -- that's sensitive to matters, you might not even want to stay to hear what this criminal -- this thing is about. It might be disturbing, shall I say.

I'll see you back here, though, to start this matter, hopefully, at 1:30.

COURT SECURITY OFFICER: All rise.

(Recess.)

* * * * * *

CERTIFICATION

I HEREBY CERTIFY that the foregoing is a true and correct transcript from the stenographic notes of the proceedings in the above-entitled matter to the best of my ability.

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